

**EPIDEMIOLOGIC TRANSITION AMONG A NATIVE AMERICAN COMMUNITY IN  
KANSAS DURING THE 20TH CENTURY**

By

©2014

Steve M. Corbett

Submitted to the graduate degree program in Anthropology and the Graduate Faculty of the  
University of Kansas in partial fulfillment of the requirements for the degree of Doctor of  
Philosophy.

---

Chairperson Dr. Sandra J. Gray  
Anthropology

---

Dr. Jeanne Drisko  
Integrative Medicine

---

Dr. Kelly Kindscher  
Environmental Studies

---

Dr. James H. Mielke  
Anthropology

---

Dr. Devon A. Mihesuah  
Humanities and Western Civilization

---

Dr. Donald D. Stull  
Anthropology

Date Defended: April 11<sup>th</sup>, 2014

The Dissertation Committee for Steve M. Corbett  
certifies that this is the approved version of the following dissertation:

EPIDEMIOLOGIC TRANSITION AMONG A NATIVE AMERICAN COMMUNITY IN  
KANSAS DURING THE 20TH CENTURY

---

Chairperson Dr. Sandra J. Gray

Date approved: April 21<sup>st</sup>, 2014

# **EPIDEMIOLOGIC TRANSITION AMONG A NATIVE AMERICAN COMMUNITY IN KANSAS DURING THE 20TH CENTURY**

## **Abstract**

This dissertation is an examination of the mortality patterns of Native Americans during the 20<sup>th</sup> century, particularly the Prairie Band Potawatomi Reservation and surrounding communities in Kansas. Shifts in mortality patterns are the focus of epidemiologic transition theory, which describes the patterns as models that are specific to populations and due to a number of disease determinants. This examination describes the epidemiologic transition among Native Americans generally and the Prairie Band Potawatomi community specifically, and compares that pattern to the classical (Western) model that characterizes the United States.

The mortality pattern of Native Americans featured high rates of infectious diseases during the first half of the 20<sup>th</sup> century, followed by a dramatic rise in chronic diseases during the second half of the century. Subsequent high rates of illnesses related to metabolic disorders led to the development of hypotheses to account for the rise of these conditions during the modern era. The thrifty gene hypothesis attempts to explain obesity-related illnesses as a genetic maladaptation to a modern lifestyle. The New World syndrome concept applies the thrifty gene hypothesis to indigenous American populations in particular. Both ideas have been prominent explanations for the current epidemiology of Native Americans.

The epidemiologic transition among the Prairie Band Potawatomi community and other Native Americans differs markedly from that experienced by the general U.S. population in being delayed and featuring higher death rates, a dramatic increase in certain chronic diseases, and a high rate of deaths due to accidents. The Prairie Band community differed from other

Native American groups in having higher death rates due to heart disease and cancer and lower death rates due to suicides and homicides.

The results of this analysis call into question the preeminence of New World syndrome and “thrifty genes” as the primary causes of high rates of obesity, diabetes, and other metabolic conditions, and suggest research into other areas that may better account for the unique disease patterns among Native Americans. Metabolic diseases such as diabetes result from a mosaic of causal factors, which now include epigenetic factors that provide a greater understanding of environmental causes and intergenerational trauma.

## **Acknowledgements**

There are a number of folks to whom I am indebted for helping me see this project through to completion. Acknowledgement of these individuals is not presented lightly, I greatly appreciate the support I have received from a number of individuals. First of all I would like to acknowledge the members of my committee, especially Dr. Sandra Gray for her mentorship, advocacy, and for allowing me to pursue my interests in the course of completing this dissertation. I would like to thank Dr. Jim Mielke and Dr. Don Stull for resources and helpful comments on my field statements. In addition I am thankful to Dr. Jeanne Drisko, Dr. Kelly Kindscher, and Dr. Devon Mihesuah for providing me with financial support and collaboration opportunities. Of course I thank all of my committee members for agreeing to serve, reading my dissertation, and providing helpful feedback. Additionally I want to thank Dr. Linda Redford for initiating the work with the Prairie Band Potawatomi and Kickapoo tribes. And thanks to Paula Smith for her help in preparing me for the defense.

During the course of this project I worked with a number of people from the Prairie Band Potawatomi community and clinic. There are so many good people on the reservation that I worked with over the years. In particular I want to acknowledge Sebe Masquat, Roy Spoonhunter, Richard Pruiksma, and Randy Mitchell for their valuable work on the Diabetes Program, and Sogi Leclere, John Holtz, Tim Mendez, Mina Neubarger, Sarah Price, Angela Emert, Mary Shopteese, Dawn Matchie, Jeanette Little Sun, Jenell Kern, Laura Thackery, and Nancy Stegeman for their assistance to the grant programs at the Prairie Band Potawatomi Health Center. I want to acknowledge the Diabetes Prevention Program participants, as well as community members and others involved with the Reclaiming a Health Past, Honoring the Gift of Heart Health, and Working Together for Balance programs. I would also like to thank the

staff and students of the Prairie Band Potawatomi Language and Cultural Department for allowing me to participate and accepting me as a full part of the program. I especially want to acknowledge Mike “Ne-se-ka” Jensen and Jim “Na-nim-nuk-shkuk” Mckinney as friends of mine, both of whom I miss greatly and appreciate having the opportunity to know.

I owe a great debt of gratitude to my family for their support and encouragement during this process. I especially want to thank Lisa Spangler, and my mother, Sondra Kemberling, for their support during this long process. I also want to thank my grandparents, Jean and ‘Okie’ Snedeger, for providing me with general guidance on how to live one’s life.

## Contents

List of Figures .....	xi
List of Tables .....	xiv
Chapter 1: Introduction .....	1
The Problem.....	1
Background .....	2
Dissertation Overview .....	5
Chapter 1 References Cited .....	7
Chapter 2: The Prairie Band Potawatomi Community .....	9
Potawatomi History .....	9
Origins of the Prairie Band Potawatomi .....	19
Fighting to Maintain Tribal Integrity.....	28
Self-Determination.....	36
Chapter 2 References Cited .....	39
Chapter 3: Epidemiologic Transition.....	42
The Age of Pestilence and Famine .....	46
The Age of Receding Pandemics.....	47
The Age of Degenerative and Man-Made Diseases .....	49
Causes of Disease Transition.....	52
Living Standards and Nutritional Intake .....	53
Public Health and Medical Services .....	55
Personal and Household Hygiene .....	58
Immunization .....	60
Changes in Disease Virulence.....	61
Increase in Chronic Disease.....	62
Variation in the Epidemiologic Transition .....	63
Socioeconomic Differences .....	63
Variation by Race, Ethnicity, and Gender .....	67
Genetic Influences on Variation in Mortality and Disease susceptibility within Populations .....	70
Epidemiologic Transition among Native American Groups .....	71

Conclusion .....	76
Chapter 3 References Cited .....	76
Chapter 4: Theoretical Roles of Biology and Society in the Epidemiologic Transition among Native Americans.....	84
Introduction.....	84
Biological Explanations .....	84
The Thrifty Gene Hypothesis.....	84
Insulin Resistance .....	87
Drifty Gene Hypothesis .....	91
Thrifty Phenotype Hypothesis .....	92
Epigenetics .....	93
Summary .....	95
Summary and Conclusion .....	123
Chapter 4 References Cited .....	124
Chapter 5: The Epidemiologic Transition in Native American Populations in the 20 <sup>th</sup> Century	132
Native American Health Data.....	132
Trends in Native American Health .....	134
Late 19 <sup>th</sup> and Early 20 <sup>th</sup> Centuries.....	134
1920s and 1930s.....	135
1940s and 1950s.....	139
1960s and 1970s.....	142
1980s to the 21 <sup>st</sup> Century .....	149
Discussion.....	153
Health Improvements in Native American populations.....	153
Epidemiologic Transition.....	155
Modern Health Characteristics .....	158
Diabetes.....	158
Cardiovascular Disease.....	161
Cancer .....	162
Social Pathologies .....	163
Causes of the Observed Pattern .....	165



Obesity .....	165
Diet.....	166
Activity .....	171
Genetics.....	172
Economic Status.....	180
Historical Trauma and Structural Violence .....	182
Chapter 5 References Cited .....	184
Chapter 6: Health and Disease among Prairie Band Potawatomi in the early 20th Century. Part I.	
The Reports of the Potawatomi Agency .....	199
Methods .....	199
Data .....	199
Results.....	202
Letters of the Potawatomi Agency, 1899-1933 .....	202
General Survey of the Potawatomi Sub Agency, Haskell Institute, April 1931 .....	205
Health of Potawatomi in 1935: The Potawatomi Agency Report.....	211
Discussion.....	212
Diet.....	213
Activity .....	217
Economic Status.....	219
Conclusion .....	220
Chapter 6 References Cited .....	221
Chapter 7: Patterns of health and disease among Prairie Band Potawatomi in the early 20th Century. Part II. The Indian Health Survey, ca. 1928.....	
Introduction.....	223
Data .....	224
Chapter 7 References Cited .....	256
Chapter 8: Mortality 1974 – 2004.....	
Introduction.....	258
Methods .....	258
Data .....	258
Statistical methods .....	259

Results.....	260
Discussion .....	278
Conclusions.....	286
Chapter 8 References Cited .....	288
Chapter 9: Conclusions .....	292
Modernization.....	295
Current Epidemiology.....	299
Transgenerational Effects .....	304
Summary and Closing.....	306
Further Research .....	315
Chapter 9 References Cited .....	318
Appendix A.....	324
Appendix B.....	326
Appendix B References Cited.....	328

## List of Figures

FIGURE 1-1. Turnbull House on the Prairie Band Potawatomi Reservation. ....	3
FIGURE 2-1. Westward migration of the Prairie Band Potawatomi.....	10
FIGURE 2-2. Door Peninsula and Green Bay in Wisconsin.....	11
FIGURE 2-3. A protohistoric Potawatomi village scene.....	13
FIGURE 2-4. Shabbona, an influential leader of the Potawatomi.....	16
FIGURE 2-5. Postcard depicting the signing of the Treaty of Chicago .....	20
FIGURE 2-6. The Platte Region of Missouri .....	21
FIGURE 2-7. Map of Emigrant Indian lands, including Potawatomi lands near Council Bluffs, north of the Platte Region .....	23
FIGURE 2-8. Gunn and Mitchell’s map showing the Potawatomi National Reserve in Kansas	24
FIGURE 2-9. The reduced Prairie Band Potawatomi Reservation in northeastern Kansas .....	28
FIGURE 3-1. Model of the Age of Degenerative and Man-Made Diseases of the Epidemiologic Transition in the U.S. ....	50
FIGURE 5-1. Causes of death by percent for the U.S. total population and Native Americans, 1934. Ill-defined causes result from a lack of accurate diagnosis .....	138
FIGURE 5-2. Declines in infant mortality since 1955 among Native Americans and the total U.S. ....	154
FIGURE 5-3. Mortality transition among Native Americans in the U.S. Created using source data from the years 1921, 1925, 1929, 1935, 1940, 1945, 1950, 1955, 1961, 1967, 1971, 1975, 1982, 1991, and 2000.....	155
FIGURE 5-4. Traditional wild rice harvest using birch bark canoes .....	167
FIGURE 6-1. Expansion of the registration areas for deaths .....	200
FIGURE 6-2. Indian blood quantum among adult Potawatomi, 1931. ....	205
FIGURE 6-3. Indian blood quantum among Potawatomi children, 1931. ....	206
FIGURE 6-4. (Left) Potawatomi men butchering a deer on the Prairie Band reservation, 1930s; (Right) 1930s view of a Prairie Band Potawatomi wood frame building and a garden plot with an arched branch framework .....	215
FIGURE 6-5. Prairie Band elder identifies milkweed ( <i>nInwezhe’k</i> ), a traditional, wild plant food. .....	216

FIGURE 6-6. (Top) Potawatomi man collecting tree bark, circa 1930s; (Bottom) Potawatomi woman carrying reeds for baskets and mats .....	218
FIGURE 7-1. Age distribution among the Prairie Band Potawatomi in 1928. ....	229
FIGURE 7-2. Population pyramid for the Prairie Band Potawatomi in 1928. ....	230
FIGURE 7-3. Age distributions of deaths where age is reported. ....	234
FIGURE 7-4. Potawatomi woman with wild fruits, probably persimmons, Prairie Band reservation, 1930s .....	253
FIGURE 8-1. Mean age at death among females in Jackson and Shawnee counties, 1974-2004, with linear trend line. ....	261
FIGURE 8-2. Mean age at death among males in Jackson and Shawnee counties, 1974-2004, with linear trend line. ....	261
FIGURE 8-3. Population pyramids showing the age distributions of Native Americans in Jackson and Shawnee counties, 1974, 1984, 1994, and 2004.....	262
FIGURE 8-4. Age adjusted death rates per 100,000 for all causes of mortality among Native Americans in Jackson and Shawnee Counties, with linear trend line.....	267
FIGURE 8-5. Native Americans in Jackson and Shawnee counties vs. US age-adjusted death rates per 100,000 population.....	268
FIGURE 8-6. Trend lines for Native Americans in Jackson and Shawnee counties vs. US age-adjusted death rates per 100,000 population.....	268
FIGURE 8-7. Age adjusted death rates per 100,000 for heart disease among Native Americans in Jackson and Shawnee Counties, with linear trend line.....	269
FIGURE 8-8. Age adjusted death rates per 100,000 for cancers among Native Americans in Jackson and Shawnee Counties, with linear trend line.....	269
FIGURE 8-9. Age adjusted death rates per 100,000 for strokes among Native Americans in Jackson and Shawnee Counties, with linear trend line.....	270
FIGURE 8-10. Age adjusted death rates per 100,000 for accidents among Native Americans in Jackson and Shawnee Counties, with linear trend line.....	270
FIGURE 8-11. Age adjusted death rates per 100,000 for diabetes among Native Americans in Jackson and Shawnee Counties, with linear trend line.....	271
FIGURE 8-12. Age adjusted death rates per 100,000 for liver diseases among Native Americans in Jackson and Shawnee Counties, with linear trend line.....	271

FIGURE 8-13. Age adjusted death rates per 100,000 for influenza and pneumonia among Native Americans in Jackson and Shawnee Counties, with linear trend line.....	272
FIGURE 8-14. Age adjusted death rates per 100,000 for COPD among Native Americans in Jackson and Shawnee Counties, with linear trend line. ....	272
FIGURE 8-15. Age adjusted death rates per 100,000 for kidney disease among Native Americans in Jackson and Shawnee Counties, with linear trend line.....	273
FIGURE 8-16. Age adjusted death rates per 100,000 for Alzheimer’s disease among Native Americans in Jackson and Shawnee Counties, with linear trend line.....	273
FIGURE 8-17. Age adjusted death rates per 100,000 for suicides among Native Americans in Jackson and Shawnee Counties, with linear trend line. ....	274
FIGURE 8-18. Age adjusted death rates per 100,000 for homicides among Native Americans in Jackson and Shawnee Counties, with linear trend line. ....	274
FIGURE 9-1. Mortality transition among the Prairie Band community. ....	293
FIGURE 9-2. Food vendors at a Prairie Band Potawatomi Pow-Wow, 2009. Foods being sold include typical fair and carnival types of foods such as funnel cakes, corn dogs, pork tenderloin, fried chicken fingers, Indian tacos, kettle corn, and curly fries. ....	300

## List of Tables

TABLE 4-1. Labor force and unemployment statistics for Native Americans and U.S. whites, 1950 – 2000, by sex .....	121
TABLE 5-1. Percentage of total deaths (crude deaths) by age and race/ethnicity, United States: 1934.....	139
TABLE 5-2. Death rates per 1,000 population for selected races, by sex, 1940, 1950, 1960...	141
TABLE 5-4. Cause-specific neonatal and post-neonatal mortality rates per 1,000 aged <1 year among Native Americans compared to the general U.S. population, 1960-1966.....	144
TABLE 5-5. Infant mortality rates (per 1,000 live births), 1966-1971 .....	145
TABLE 5-6. Causes of infant deaths by percent, 1969 .....	145
TABLE 5-7. Deaths due to tuberculosis, 1955-1971.....	146
TABLE 5-8. Comparison of Maternal mortality rates per 100,000 live births among American Indians and the general U.S. Population, 1958-1967.....	147
TABLE 5-9. Comparison of life expectancy at birth, at 1 year and at age 5 for American Indians and the general U.S. population.....	147
TABLE 5-10. Changes in crude cause-specific death rates (per 100,000) among American Indians compared with the total U.S. population.....	149
TABLE 5-11. Changes in crude death rates per 100,000 for Indians and the total U.S. population for selected causes – 1955, 1960, 1965-1967. ....	150
TABLE 5-12. Number of deaths and crude death rates (per 100,000) for Indians of the IHS Kansas Service Unit, 1972-1974, 1975-1977 .....	150
TABLE 5-13. Life expectancy at birth among American Indians, 1940 – 1980.....	152
TABLE 5-14. Causes of Mortality by percent and by race, 2000 .....	153
TABLE 5-15. Causes of Mortality by percent among Native Americans in 1945 and 1950....	157
TABLE 5-16. Gene regions with loci associated with diabetes and metabolic syndrome and allele frequencies for selected populations .....	175
TABLE 5-17. Overview of the physiologic impact of loci associated with diabetes-related traits at genome-wide significance.....	176
TABLE 5-18. Annual income of selected racial groups in 1970.....	181
TABLE 6-1. Illnesses diagnosed by Special Physician J.L. Goodwin among 43 Prairie Band Potawatomi tribal members in March 1928.....	204

TABLE 6-2. Personal Property owned by Potawatomi households as reported by the 1931 General Survey.....	207
TABLE 6-3. Medical cases reported in the 1931 General Survey. ....	208
TABLE 6-4. Causes of death among Potawatomi at the Potawatomi Agency, 1924-1933. ....	208
TABLE 6-5. Incidence of specific health conditions, Prairie Band Potawatomi Reservation, 1935.....	209
TABLE 6-6. Health care and selected health statistics for the Prairie Band Potawatomi reservation, 1935.....	210
TABLE 6-7. Causes of death on the Prairie Band Potawatomi Reservation, 1935.....	211
TABLE 6-8. General Purchase Order of the Potawatomi Agency in 1932. ....	214
TABLE 6-9. Top-ten causes of death in the U.S., all populations, 1935 .....	221
TABLE 7-1. General demographics of the people surveyed in 1928.....	229
TABLE 7-2. Occupations in 1928. ....	231
TABLE 7-3. Income groups, 1928, based on surveyors' summaries .....	231
TABLE 7-4. Sanitary condition of housing in 1928.....	232
TABLE 7-5. Household water sources. ....	232
TABLE 7-6. Toilet facilities in 1928.....	233
TABLE 7-7. Five most often reported causes of death .....	234
TABLE 7-8. Five most often reported health problems in 1928. ....	236
TABLE 7-9. Most common serious illnesses among households in the five previous years. ....	237
TABLE 7-10. Health Care Services utilized in 1928. ....	237
TABLE 7-12. Macronutrient intake in 1928.....	239
TABLE 7-13. Most significant correlations with disease variables. ....	240
TABLE 7-14. Most significant correlations with biological variables.....	241
TABLE 7-15. Most significant correlations with environmental variables.....	242
TABLE 7-16. Most significant correlations with dietary variables.....	243
TABLE 7-17. Factor analysis of variables from 1928. Positive loadings shown in green, negative loadings in yellow, and the highest communalities in blue.....	244
TABLE 7-18. Partial least squares analysis with ANOVA of specific variables, using factor scores from the diet, blood quantum, and environment factors. ....	244

TABLE 7-19. Regression coefficients for variables significantly predicted by the PLS model.	245
TABLE 7-20. Top 15 causes of death in Kansas, 1928.....	256
TABLE 8-1. Age-adjusted causes of death by percent among the Native Americans in Jackson and Shawnee counties 1974, 1980, 1985, 1990, 1995, 2000, 2004, and ranked by mortality rate, 1974-2004. ....	263
TABLE 8-2. Comparison of change in death rates from 1974 – 2004 for Native Americans in Jackson and Shawnee counties and for all Kansas Native Americans. Numbers are based on linear trend lines on age-adjusted death rates per 100,000 population. ....	264
TABLE 8-3. “Recent” age-adjusted causes of death by percent among Native Americans in Jackson and Shawnee counties, 1990-2004. ....	265
TABLE 8-4. Cancer deaths by type, Native Americans in Jackson and Shawnee counties, 1990-2004.....	266
TABLE 8-5. Comparison of crude and age-adjusted mortality rates for Native Americans in Jackson and Shawnee counties from 1996 to 2004; age adjusted mortality rates for Native Americans (AI/AN), 2004 to 2006 and age-adjusted rates for the U.S in 2000. Tests of significance compare the JK/SH 1996-2004 age-adjusted death rates with those of the U.S. in 2000 (standard population) .....	275
TABLE 8-6. Crude mortality rankings from 2002 for racial groups (Anderson 2002), compared to rankings for Native Americans in Jackson and Shawnee counties for data from 1990-2004.	276
TABLE 8-7. Cancer deaths by type for Native Americans in Jackson and Shawnee counties, 1990-2004, compared to U.S. rates, 2005.....	277
TABLE 8-8. Comparison of crude death rates by cause between Native Americans in Jackson and Shawnee counties, 1990-2004; and the total American Indian population, 2000.....	278
TABLE 8-9. Native American access to diabetes treatment and prevention services after the implementation of the SDPI.....	282
TABLE 9-1. Total number of Indian deaths in Kansas, 1960-1971 .....	292



# **Chapter 1: Introduction**

## **The Problem**

Epidemiologic transition theory provides an explanation of how diseases that cause death vary through time, and suggests populations follow specific patterns of disease transition related to sociohistorical circumstances (Omran 1972). How does the pattern of epidemiologic transition differ among Native Americans and the Prairie Band Potawatomi community when compared to other U.S. populations? What are the causes of the observed pattern?

Near epidemic levels of obesity-related chronic diseases now affect Native Americans. Ironically, there was a time in the 20<sup>th</sup> century when Native Americans were thought to be immune to some of these chronic diseases, particularly diabetes (Chase 1937). Why such a radical change in a relatively short period of time? Geneticist James Neel (1962) postulated that genes adapted to the hunter-gatherer subsistence pattern of the human past conferred a tendency among some individuals to store fat more efficiently as a reserve fuel source for times of food scarcity. During the modern era, with little or no risk of food scarcity, individuals carrying these “thrifty genes” tend toward obesity and associated health conditions. The assortment of obesity-related metabolic conditions disproportionately afflicting Native Americans led to the “New World Syndrome” concept (Weiss, Ferrell, and Hanis 1984), which applied Neel’s theory of thrifty genes to the particular circumstances of Native American populations. Do these concepts adequately explain the epidemiologic transition of the Potawatomi and other Native American groups? Why have certain metabolic and chronic diseases become so prevalent among Indian populations such as those living on the Prairie Band Potawatomi Nation Reservation and surrounding towns that compose the Prairie Band Potawatomi (PBP) community?

This dissertation addresses the questions above, and in so doing will:

- establish the epidemiologic transition of Prairie Band Potawatomi and other Native Americans;
- evaluate the thrifty gene hypothesis and New World syndrome as explanations of the current Native American epidemiology;
- provide an alternative explanation for the epidemiologic transition as experienced by the Prairie Band Potawatomi and other Native Americans.

## **Background**

I began working with the Prairie Band Potawatomi Nation in 2002 as a senior research associate at the Landon Center on Aging at the University of Kansas Medical Center (KUMC), working for the Kansas Rural Interdisciplinary Training Program (KS-RIT). The program was funded through a Quentin N. Burdick Rural Health Interdisciplinary Training grant from the Bureau of Health Professions, Health Resources and Services Administration (HRSA), Department of Health and Human Services (DHHS). This work involved coordinating and supervising KUMC health professions students who would live and work on the Prairie Band and Kickapoo reservations as a part of providing the students with a rural, cross-cultural experience. I coordinated the students' efforts, providing them with assignments at the various tribal organizations, primarily the PBP tribe's clinic. As a way of providing cultural competency education, I also provided the students with historical and cultural information on the tribes and brought in tribal members to speak to the students about cultural traditions. During the summer months from 2003 – 2006, I regularly lived on the Prairie Band Reservation with the students at the Turnbull House, provided by the tribe (Figure 1-1). The KS-RIT program also incorporated health screenings for reservation residents throughout the year, providing blood glucose, cholesterol, blood pressure, and other health parameter checks. These screenings included data

collection and analysis that first indicated to me the severity of the diabetes epidemic among this community. In the process of conducting background research on the health of the PBP in the past for comparison with the screening results, I came across the *Records of the Bureau of Indian Affairs (BIA), Record Group 75, 1793-1989*; specifically, the *Records of the Health Division, 75.14.4*, which included information on the Prairie Band.

In 2006 I went to work directly for the tribe as the diabetes project manager at the Prairie Band Potawatomi Health Center. Primary duties of the position included coordinating grant funded initiatives aimed at reducing the incidence and impact of diabetes. The initial grants for the program consisted of the Special Diabetes Program for Indians (SDPI) Community



**FIGURE 1-1. Turnbull House on the Prairie Band Potawatomi Reservation.**

Directed grant and Diabetes Prevention Program. These programs focused on the prevention of diabetes as well as the prevention of complications from the disease among diagnosed patients. The program was expanded to include other chronic conditions after I was awarded grants for preventing hypertension and heart disease in 2007 and 2008, respectively. Two small grants from the Kansas Department of Health and Environment allowed for the establishment of Chronic Disease Electronic Management Systems (CDEMS) for diabetes and hypertension. CDEMS is a software application designed to assist medical providers and management in tracking the care of patients with chronic health conditions.

In 2008 I received a grant from the Centers for Disease Control and Prevention titled “Using Traditional Foods and Sustainable Ecological Approaches to Prevent Diabetes among American Indian/Alaska Native Communities.” A focus of the grant was to establish the timing of changes in dietary and activity patterns as a basis for returning to a time when diabetes was rare. This project provided me with the opportunity to speak with tribal elders regarding their perceptions of traditional foods and how diet, activity, and disease patterns changed over time. These elders ranged in age from the late 50s to over 100. Most were in their 70s.

Following my departure from the clinic in 2009, I continued to work with individuals and organizations on the reservation, including writing the Diabetes Prevention Program and the Potawatomi Language and Revitalization Project grants in 2010 and 2011, respectively. With so many years of involvement on the reservation, I was able to observe the dynamics of the community over an extended period of time, including the variety of perspectives among the people and swings of the political pendulum.

## **Dissertation Overview**

The first five chapters of this dissertation provide background information necessary for understanding the subjects, concepts, and circumstances referred to in the subsequent chapters and conclusion. The chapters are largely discrete and include their own methods, results, and discussion sections, where appropriate.

Chapter 2 describes the Prairie Band Potawatomi (PBP). This is primarily a historical review of the Potawatomi tribe and the development of the Prairie Band as a distinct band. This review includes a description of how the PBP came to Kansas and the tribe's relationship with the federal government. This section provides a basis for understanding PBP cultural and socioeconomic circumstances during the 20<sup>th</sup> century.

Chapter 3 presents an overview of epidemiologic transition theory as developed by Abdel Omran (1971). The theory features five propositions, three ages of mortality, and three models of variation in the pattern of disease transition. This chapter provides a basis for comparison of PBP and general Native American mortality patterns. Chapter 3 also includes a discussion of the causes of disease transition.

Chapter 4 is a discussion of the roles that biological and societal factors play in Native American epidemiologic and mortality patterns. As biological factors, the thrifty gene hypothesis and the related concept, the New World Syndrome, are presented. Descriptions of these hypotheses provide a basis for understanding prevailing explanations of Native American epidemiology. Also included are discussions of critiques of the thrifty gene hypothesis, and presentation of alternative hypotheses. These alternative hypotheses provide a foundation for the conclusions of the dissertation. Societal factors are presented within the framework of the concept of structural violence. This concept is connected to federal Indian policy through a

discussion of the history of the U.S. government's relationship to Native American tribes. This also provides a foundation for the conclusions of the dissertation. With structural violence presented as an explanation for Native American epidemiologic patterns, the dissertation becomes an application of critical medical anthropology.

Chapter 5 is a review of the available data on Native American health during the course of the 20<sup>th</sup> century. A discussion of the lack of reliable data on Native American epidemiology is included. Data and research from other investigators are presented to provide a composite view of morbidity and mortality through time. The timing of the epidemiologic transition among Native Americans to the *Age of Degenerative and Man-Made Diseases* is pinpointed. Health improvements are discussed, as are the modern health characteristics of Native Americans. The chapter concludes with a discussion of the potential causes of the current epidemiological profile of Native Americans.

Chapter 6 presents the results of the examination of the records and letters of the Potawatomi Agency. The information from this source is primarily descriptive, and is presented as such. The records cover 1899 – 1936. Most of the records detail issues regarding the administration of the Potawatomi Reservation and other sites under BIA jurisdiction. Some records discuss the health of the Potawatomi, but detailed health statistics are often lacking. The *Potawatomi Indian Agency Annual Statistical Reports on Health (1931 – 1936)* and the *Records from the General Survey of the Potawatomi Sub Agency* were the best sources for data on health conditions.

Chapter 7 is an analysis of the Potawatomi Indian Health Survey, conducted in 1928 on the reservation. Data from this source are more amenable to statistical analyses and present counts and frequencies on illnesses, deaths, blood quantum, and living conditions. Correlations,

factor analyses, and regression analyses results of relevant variables are included. The results are evaluated for the most significant contributors to the health conditions of the time and the relative progress of the Potawatomi through the epidemiologic transition.

Chapter 8 is an analysis of mortality among Native Americans in Jackson and Shawnee counties in Kansas from 1974 to 2004. Data from the Kansas Department of Health and Environment are used to determine cause-specific death rates by year and examine changes in death rates through time. These results provide additional information on the recent mortality in the region and where this places Native Americans regarding the epidemiologic transition. This chapter includes a discussion of the shifts in mortality patterns over time and the leading contributing factors. As in Chapter 6, the discussion focuses on particular aspects of epidemiology: obesity, dietary intake, activity patterns, diabetes, cardiovascular disease, cancers, social pathologies, and socioeconomics.

Chapter 9 presents the conclusions of the dissertation. This includes a description of the epidemiologic transition as experienced by the Prairie Band and Native Americans, tying together the various chapters of the dissertation, including a discussion on lingering health disparities. The thrifty gene hypothesis and New World syndrome concept are evaluated as contributions to understanding current epidemiology of Native Americans. The role of federal Indian policy in continuing health disparities is summarized and related to the PBP in particular. This leads to a discussion of the external and internal challenges faced by the tribe in elevating the tribe's health status. The chapter and dissertation conclude with suggestions for future research.

## **Chapter 1 References Cited**

Neel, J.V. 1962. Diabetes mellitus: A Thrifty Genotype Rendered Detrimental by Progress? *American Journal of Human Genetics* 14:353 -62.

Omran, A.R. 1971 The Epidemiologic Transition; A Theory of the Epidemiology of Population Change. *The Milbank Memorial Fund Quarterly* 49(4):509-538.

Weiss, K.M., Ferrell, R.E., and Hanis, C.L. 1984. A New World Syndrome of Metabolic Diseases with a Genetic and Evolutionary Basis. *Yearbook of Physical Anthropology* 27:153-178.



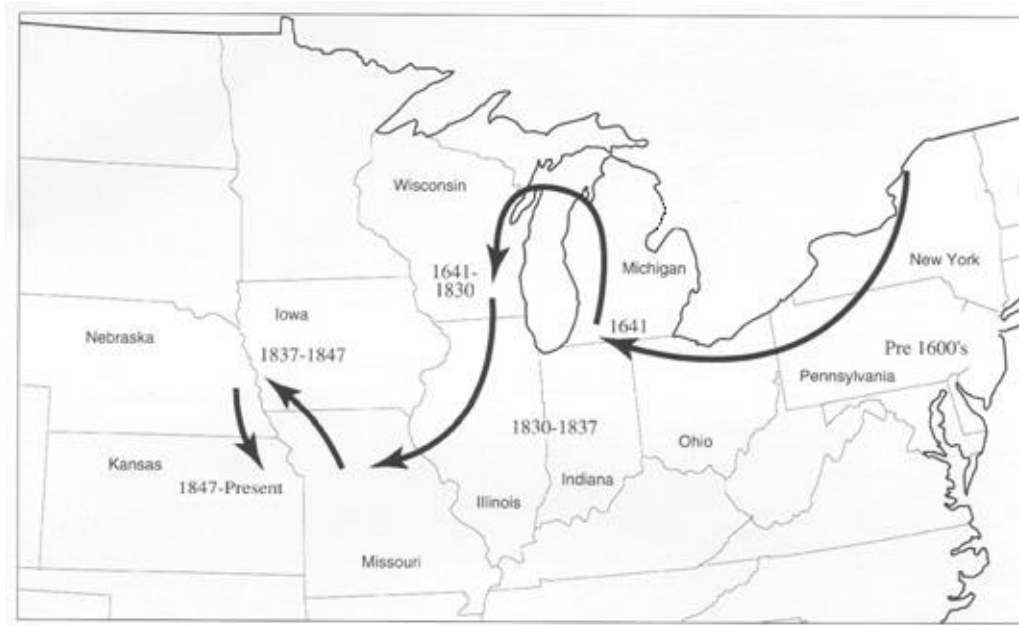
## Chapter 2: The Prairie Band Potawatomi Community

### Potawatomi History

The Prairie Band are one of several modern bands of Potawatomi, who originally lived in the Great Lakes region. They are a member of the Central Algonquian subfamily of languages, and their language is most closely associated with Odawa and Ojibwa. Prior to 1550, the Potawatomi spoke a form of Ojibwa (Clifton 1998).

The Potawatomi call themselves *Neshnabé*, “Man,” or *Neshnabék*, which means “People.” The name Potawatomi has had a number of interpretations, such as “Fire People” and “People of the Place of the Fire” (Edmunds 1978). An attempt to interpret the name using the Ojibwa – Odawa language and terms produces “by blowing” and “to make a fire” (Goddard 1972). Using the Algonquian pronunciation and interpretation produces *Bodēwahnene*, which can be interpreted as *bod* + *ewa* + *dnene* which forms “by blowing” + “to make a fire” + “people” (Clifton 1998). Oral histories of the Potawatomi, Ojibwa, and Odawa all suggest that the three groups sprang from a single population. The groups were referred to as the “three fires,” and also called the “three brothers.” Of these three brothers the Potawatomi were considered the youngest brother (Mitchell 1995).

Westward migration (Figure 2-1) in response to Iroquois expansion preceded the split into three groups, which reportedly occurred at the Straits of Mackinac. Those who moved south into Lower Michigan were the Potawatomi (Edmunds 1978). The grouping of the Potawatomi with the Odawa and Ojibwa tribes ended when the Potawatomi established themselves as an autonomous tribe, when it is said they “built a new fire.” The common interpretation of Potawatomi as “People of the Place of the Fire” may refer back to this time (Edmunds 1978; Mitchell 1995).



**FIGURE 2-1. Westward migration of the Prairie Band Potawatomi (from Mitchell 1995).**

The evidence suggests that the Potawatomi occupied the western edge of what is now Michigan, along the eastern shore of Lake Michigan. They very likely occupied this part of the Lower Michigan peninsula by about 1500 AD. The earliest historical evidence of the existence of the Potawatomi dates to 1634, in a meeting with Jean Nicolet, a French explorer. The meeting reportedly occurred at Green Bay, while the Potawatomi were visiting the Winnebago. There may have been about 2,500 Potawatomi at this time. The first reliable description of the Potawatomi came from Claude Allouez, a Jesuit Missionary, in 1667 and 1668. By this time the Potawatomi had moved to eastern Wisconsin, occupying the Door Peninsula in Lake Michigan (Edmunds 1978).

Potawatomi subsistence consisted of gathering wild vegetable foods, local pedestrian hunting, the use of birch bark canoes for traveling and hunting over longer distances as well as



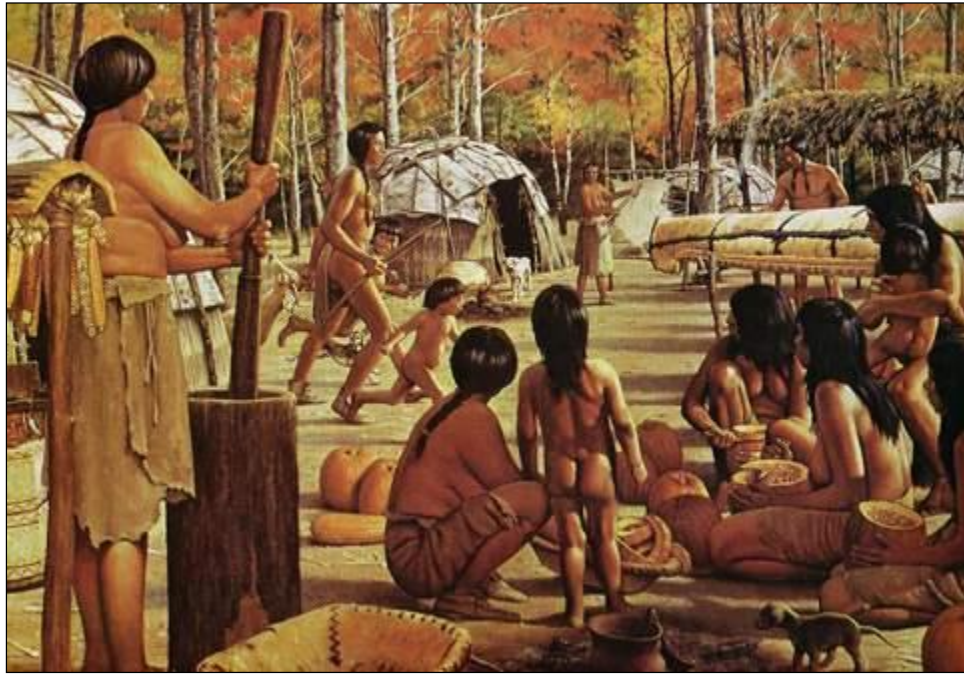
**FIGURE 2-2. Door Peninsula and Green Bay in Wisconsin. (Courtesy U.S. Census Bureau)**

for fishing, and shoreline fishing using traps and weirs. They also grew crops, most notably corn, beans, and squash. The most often exploited animals included deer and elk, as well as opossum, raccoon, rabbit, turkey, muskrat, buffalo, turtles, black bears, and beaver. The most commonly exploited fish were whitefish, herring, trout, pike, perch, sturgeon, suckers, burbot, catfish, and white bass. Wild gathered foods included acorns, walnuts, plums, butternuts, wild rice, berries, white water lily, and maple sugar (Clifton 1998).

The Potawatomi became involved in the fur trade shortly after their meeting with Jean Nicolet. The Indian tribes soon wanted European trade goods, initially steel tools such as knives, awls, and axes; and also cloth. Competition among the tribes to monopolize trade with the Europeans became fierce, and eventually led to conflict and intergroup warfare. Due to attacks from the Iroquoian Neutrals, by 1641 the Potawatomi had moved out of their Michigan homeland northward through the Upper Michigan peninsula, and then southwest to the Green Bay region (Figure 2-2) of Wisconsin (Mitchell 1995).

In response to the militarism of the Iroquois League from the east, several other Algonquian tribes gathered with the Potawatomi at Green Bay to form an alliance to repel the eastern tribes. The Potawatomi named this gathering place *Mechingan*, after the term *Mitchigami*, meaning “great lake.” The Algonquians successfully repelled the invaders, thus opening up the region to an expansion of the fur trade. By 1658, the Potawatomi were well established at Green Bay, and in a position to establish a major trade relationship with the French (Edmunds 1978).

In 1668, the Potawatomi convinced influential French trader Nicholas Perrot to establish a trade relationship with them. With this relationship the Potawatomi aimed to rival the then dominant Odawa as fur trade middlemen. By 1699 the Potawatomi villages had spread south from Green Bay to Milwaukee and were also expanding along the St. Joseph River in southern Michigan. A 1701 peace treaty with the Iroquois allowed the Potawatomi to become a principal tribe in the French fur trade in the western Great Lakes. After the founding of Fort Pontchartrain at Detroit, the French encouraged the Potawatomi to settle in the area (Edmunds 1978). Potawatomi tribal culture and society remained relatively intact during the era of their association with the French (Figure 2-3). The tribe actually thrived politically, and came to dominate many of its neighboring tribes. Throughout the 1730s the Potawatomi further demonstrated their loyalty to the French by using both warfare and diplomacy to suppress the activities of other tribes that could jeopardize their trade monopoly. After dominating the fur trade and surrounding tribes for 50 years, they began expanding with a few other tribes into the territories of their defeated and remnant rivals, such as the Illinois. Potawatomi were settling along the Chicago River by 1751, and Milwaukee and Chicago had become major Potawatomi settlements by 1761 (Edmunds 1978).



**FIGURE 2-3. A protohistoric Potawatomi village scene. (Courtesy Native American Encyclopedia)**

Starting in 1754, the Potawatomi sided with the French against the British in the French and Indian War. As a result they participated in the infamous siege of Fort Henry in 1757, and suffered from the ensuing smallpox outbreak that infected many of the participating tribes, thus limiting their involvement again until 1760. With the defeat of the French and the fall of Montreal to the British, the French and Indian War came to an end, as did the long and lucrative relationship between the Potawatomi and the French. The British wished to maintain the fur trade, establish a market for British goods, and limit the expansion of their American colonies. Just as Pontiac's War (1763-1766) erupted, the British aimed to achieve these objectives by issuing the Proclamation of 1763, which established a boundary between the English colonists and the Indians to the west. The proclamation line, which was drawn along the Appalachian

Mountains, also established an Indian Reserve between the English colonists and Spanish territories (Edmunds 1978).

The American Revolution was at first seen by the Potawatomi and many other Indian tribes as a dispute between family members that held little interest to tribal leaders. Nonetheless the vast majority of Native Americans at the western edge of the colonies distrusted the Americans and supported the British cause. Eventually the British persuaded many Potawatomi to join them. At the conclusion of the war, after the signing of the Treaty of Paris (1783), the British all but abandoned their Indian allies as the Americans took possession of all land west of the 1763 proclamation line east of the Mississippi and north of Florida. (Calloway 1995; Mitchell 1995).

The Potawatomi joined the Western Confederacy in 1785 during the Northwest Indian War. The decisive Battle of Fallen Timbers and subsequent British rejection of the Indians at Fort Miami in 1794 indicated to the Indians of the Western Confederacy that the U.S. had become the primary force in the region. The Greenville Treaty of August 1795 ended the Northwest Indian War and established a treaty of peace between the U.S. and the tribes of the Western Confederacy. One-quarter of the Indian signatories to the treaty were Potawatomi, indicating their numbers and power at the time. A condition of the treaty was that the Indians had to concede that they were now under the guardianship of the U.S., which had the sole right to negotiate for Indian lands in future treaties. In addition to establishing peace and releasing prisoners, the treaty ceded most of the modern state of Ohio, as well as tracts that would become Chicago and Detroit, and designated specific regions for Indian lands (Edmunds 1978).

The fiduciary responsibilities encoded in the trust relationship between Indians and the new American government established that the government must act in the best interests of the

Indian beneficiaries. This fiduciary principle was established in 1787 by the Northwest Ordinance, which states: "The utmost good faith shall always be observed towards the Indians; their land and property shall never be taken without their consent; and, in their property, rights, and liberty, they shall never be invaded or disturbed, unless in just and lawful wars authorized by Congress." In reality, the Northwest Ordinance primarily served to open up the region to American settlement, eventually creating the states of Ohio, Michigan, Indiana, Illinois, and Wisconsin (Stull 1984; Tyler 1973). By the end of the 1700s, Potawatomi villages were scattered throughout Michigan, Wisconsin, Indiana, and Illinois.

During the War of 1812, the U.S. hoped to keep the Indian tribes neutral and uninvolved, but the British succeeded in enlisting their old Indian allies against the Americans. The Potawatomi mostly sided with the British and participated in the capture of Forts Mackinac and Dearborn. Many Potawatomi joined Tecumseh, who also was allied with the British. Winamek and Shabbona (Figure 2-4) were among the Potawatomi chiefs to support Tecumseh. Though actually an Odawa, Shabbona had married into the Potawatomi and eventually wielded much influence in the tribe (Edmunds 1978). His legacy with the tribe would continue to be significant into the 21<sup>st</sup> century.

The Potawatomi warriors found it difficult to engage in a protracted war due to their subsistence duties back home, and by 1813 many had returned to their villages. After Tecumseh was killed at the Battle of the Thames at Moraviantown (1813), Potawatomi involvement in the War of 1812 began to diminish. In October of 1813, the Potawatomi were offered an armistice by the U.S. government (Edmunds 1978).



**FIGURE 2-4. Shabbona, an influential leader of the Potawatomi. (Courtesy Northern Illinois University)**

Prior to 1815, the Indian tribes could negotiate treaties from positions of relative power, being militarily strong and having allies such as the British. But after the British defeat in the War of 1812, Indian power diminished significantly. The U.S. pushed for treaties in its own best interests, and employed trickery, dishonesty, bribery, and coercion to get tribes to sign (Stull 1984; Tyler 1973).

The years immediately following their exit from the War of 1812 were difficult for the Potawatomi. A split developed in the tribe, with some leaders (*okamek*) favoring a peaceful coexistence with the U.S., while other leaders still wanted to resist the Americans and help the British. British supplies to the tribe were dwindling, however, as was Potawatomi support for the British. In 1815 seven Indian leaders signed the treaty of peace at Portage des Sioux, officially ending the War of 1812 for the Potawatomi. In 1816 tribal leaders signed the Treaty of



St. Louis, notable in being the first treaty in which the U.S. government purposely bargained with only a portion of a tribe as a way of dividing their power and interests. This would eventually lead to use of the “band” concept to characterize the Potawatomi and other tribes. Within thirty years of the Greenville Treaty of 1795, traditional Potawatomi sociopolitical structure had splintered into separate, competing factions; Potawatomi in the southernmost range of their territory in Illinois began to be referred to as Potawatomi of the Prairie (Clifton 1998). Thus, three precedents regarding U.S./Indian relations had been set within about 20 years: imposition of U.S. guardianship; shrinkage of Indian lands and displacement of native populations; and increasing fragmentation of tribal polities.

In March of 1824 Secretary of War John C. Calhoun established an agency to oversee Indian affairs, although without congressional authorization. Former Superintendent of Indian Trade Thomas McKenney was appointed to head the bureau, which he first called the Indian Office, and later the Office of Indian Affairs (OIA), the official name of the department until 1947, when it was changed to Bureau of Indian Affairs (BIA). Not until 1832 did Congress legislate creation of the office and authorize the president to appoint a commissioner of Indian Affairs to serve under the secretary of war (Tyler 1973). The OIA was distinctive in that it was the only federal bureau accountable for all aspects of the lives of a particular group of people, until the Indian Health Service was formed under the United States Public Health Service in 1955.

The purpose of the OIA was to protect Indian resources, build and support infrastructure and services to Indians, and assist Indian tribes in maximizing their own capabilities (Stull 1984); however, non-Indian demand for and encroachment on Indian lands eventually led to the Indian Removal Act of 1830. The removal policy supported treaties that would allocate to the

Indians lands west of the Mississippi River in exchange for their lands in the east. Proponents of the removal policy argued that it was in the best interests of Indian survival. Conflict between the federal and state governments over jurisdiction of Indian affairs prompted others to see removal as a solution to this conflict, which threatened government stability prior to the Civil War (Prucha 1984).

The Cherokee attempted to challenge the Indian Removal Act, which was promoted by President Jackson and the state government of Georgia. In *Worcester v. Georgia* [31 U.S. (6 Pet.) 515, 557 (1832)], the U.S. Supreme Court decided that the Cherokee Nation was exempt from any laws of state governments that infringed on tribal sovereignty. *Worcester v. Georgia* invalidated a Georgia statute first challenged in the court case *Cherokee Nation v. Georgia* [30 U.S. (5 Pet.) 2 (1831)], the case that first described Indian tribes not as foreign nations but as “denominated domestic dependent nations.” The state statute conferred upon the state of Georgia jurisdiction over individuals living on Cherokee lands. The Supreme Court, under Chief Justice John Marshall, ruled that Indian tribes are guaranteed federal protection from interference from state governments. Although *Worcester v. Georgia* and *Cherokee Nation v. Georgia* helped to establish the trust relationship between Indian tribes and the federal government (Berry et al. 2004; Prucha 1984), President Jackson continued to pressure the eastern Indian tribes to sign treaties of removal. Such treaties were negotiated with the Five Civilized Tribes (Cherokee, Chickasaw, Choctaw, Creek and Seminole) of the southeast U.S. between 1830 and 1836. By 1840, most of the Indians of the southeast were relocated to Indian Territory in present-day Oklahoma (Prucha 1984).

Removal treaties were also negotiated with tribes remaining in the Old Northwest Territory. Whereas in the south the tribes were few, large, and had well-defined territorial

boundaries, the northeastern tribes were numerous, scattered, nomadic, and often had overlapping land claims. The process of negotiating treaties with such a diverse collection of tribes was a complex and prolonged process. Removal of some tribes, such as the Potawatomi and Winnebago, was a piecemeal process lasting several decades (Prucha 1984).

## **Origins of the Prairie Band Potawatomi**

The modern Prairie Band Potawatomi of Kansas developed from the United Bands that settled first in Missouri, and then Iowa. The Potawatomi of the United Bands came mostly from southern Wisconsin and northern Illinois. The Prairie Band became distinct in 1861 (Clifton 1998).

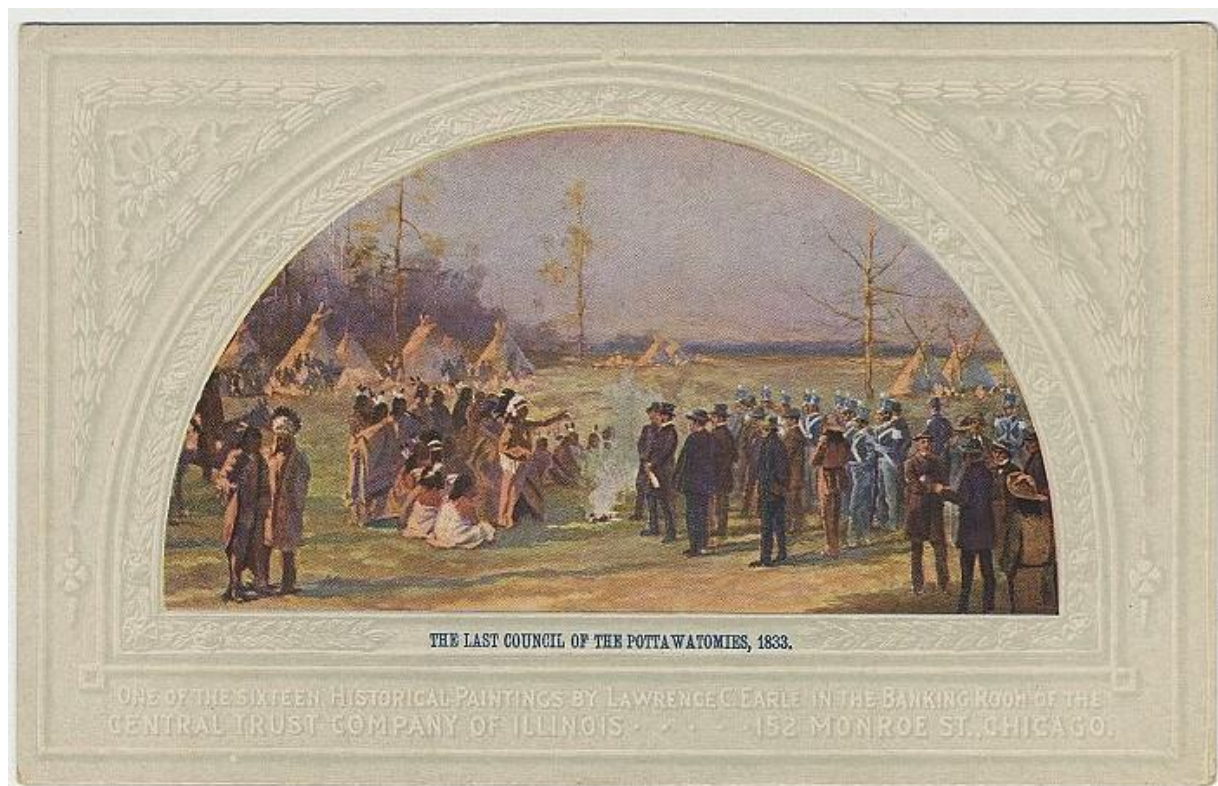
In 1827, a treaty signed at St. Joseph relocated Potawatomi from around the Detroit area to far western Michigan, away from American settlements. With the enactment of the Indian Removal Act in 1830, the tribe was aware that removal to the west was a likely prospect. Potawatomi groups argued over the terms of new treaties that might lead to removal. Treaty commissioners attempted to resolve these disputes by developing three separate treaties. The first involved the Potawatomi of southern Wisconsin and northern Illinois, referred to as the Potawatomi of the Prairie and Kankakee, and also as the United Bands, and included members of the Odawa and Ojibwa tribes. The second was with Potawatomi living along the Elkhart and Wabash rivers in northern and western Indiana. The final treaty concerned the remaining Potawatomi living in Indiana and Michigan (Edmunds 1978).

American settlers were moving into Illinois and Michigan in droves by 1833, increasing the pressure for the Potawatomi to move west (Edmunds 1978). At the time, the Potawatomi had over 50 villages in Illinois, Michigan, Wisconsin, and Indiana, all of which blocked American westward expansion (Herring 1990). U.S. influence and contact had been most intense among

the eastern Potawatomi, who had to adapt to the presence of the Europeans and make certain accommodations. The Potawatomi to the west in Wisconsin and Illinois had much less contact with and were more antagonistic toward the Americans (Clifton 1998; Mitchell 1995).

Intermarriage between French men and Potawatomi women was more common in Michigan and Indiana than in the areas further west, which led to greater assimilation among these Potawatomi into white culture. In contrast, the Wisconsin and Illinois villages attempted to preserve their traditions, and many rejected Christianity and other aspects of white society (Mitchell 1995).

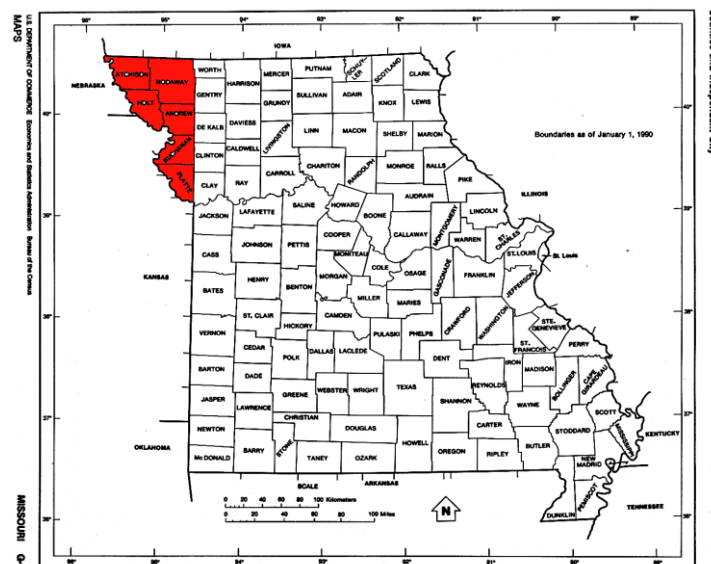
A treaty signed in Chicago in 1833 transferred a vast section of Potawatomi lands to the U.S. The Odawa, Ojibwa, and Potawatomi sold the last remaining precontact tracts of land, and the Wisconsin and Illinois Potawatomi sold the land between Milwaukee and Chicago in



**FIGURE 2-5. Postcard depicting the signing of the Treaty of Chicago. (Author's personal collection)**

exchange for promises of cash payments and tracts of land west of the Mississippi (Figure 2-5). The Chicago treaty was the last significant Indian treaty involving the lands of the Great Lakes (Edmunds 1978). The Potawatomi received five million acres in the Platte country, just east of the Missouri River (Figure 2-6). They promised to migrate to these new lands west of the Mississippi as soon as possible, abandoning all their land in Illinois once the ratification of the treaty was complete. After ratification of the treaty in 1835, the Potawatomi were given until September of 1836 to leave.

Most of the Potawatomi from northern Illinois and southern Wisconsin (United Bands) moved to the Platte region of Missouri between 1835 and 1837. Those who moved to Missouri found the land to be favorable. They described it as having fertile soils, abundant water sources, and plenty of timber. Due to these favorable conditions, however, American settlers and the Missouri government also wanted the land (Mitchell 1995), which the state of Missouri was also attempting to annex (Clifton 1998).



**FIGURE 2-6. The Platte Region of Missouri. (Courtesy Wikimedia Commons)**

State senators from Missouri refused to recognize the 1833 treaty in Chicago until the Platte region was excluded as a part of the deal. A revised treaty was approved by the U.S. Senate in 1835, and Missouri was free to annex the Platte Country. However, the region had already begun to be colonized by Potawatomi moving west (Clifton 1998), and others still living in the Great Lakes region were making plans for the move to Missouri even as the land passed out of their hands: the state of Missouri annexed the Platte Country in 1837, and the U.S. government determined that it would have to relocate all of the Potawatomi in the region (Clifton 1998; Mitchell 1995).

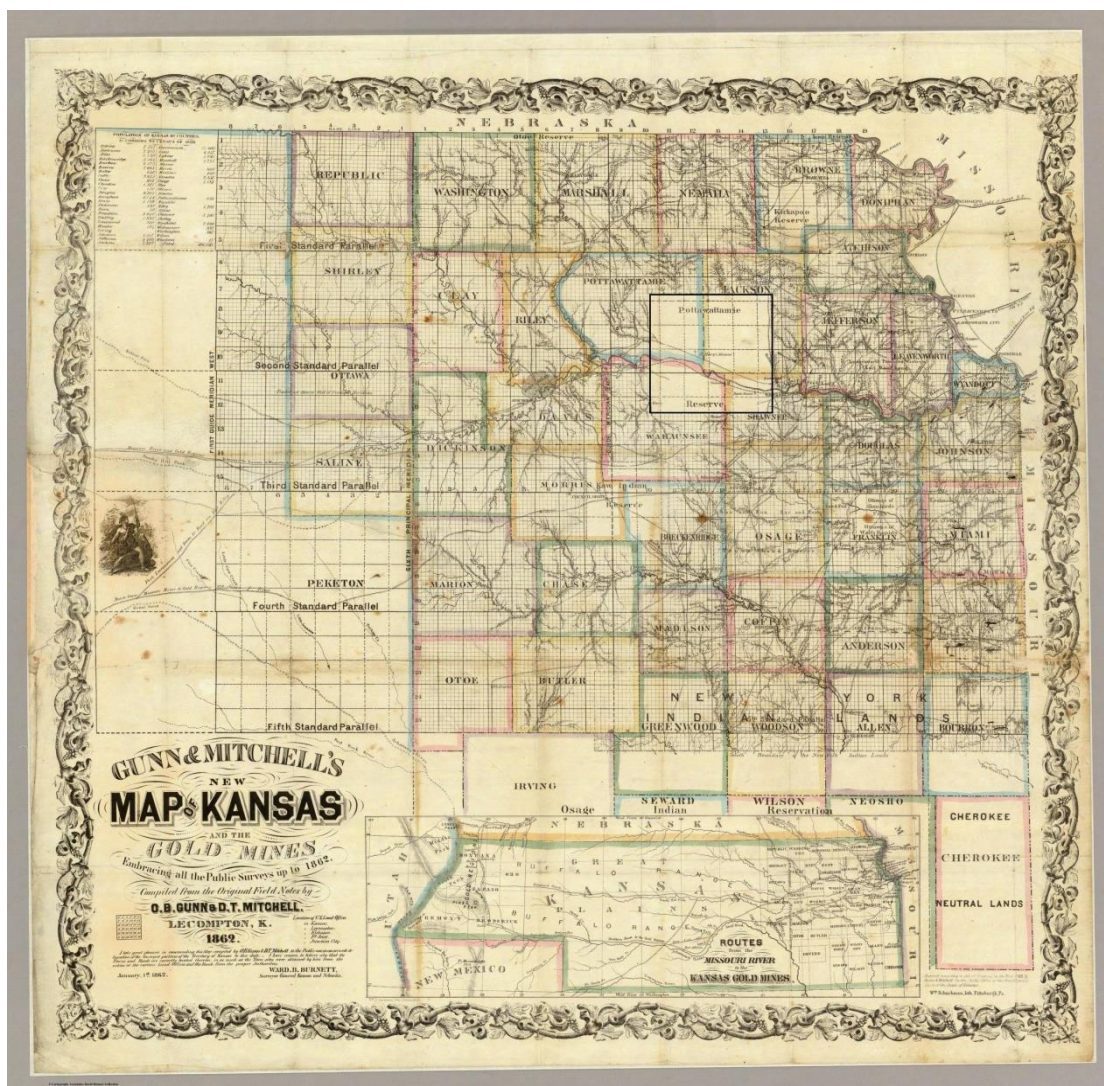
The United Bands of the Platte agreed to move north to the newly created Council Bluffs sub-agency of the Office of Indian Affairs (Figure 2-7), but the Potawatomi from Indiana and Michigan, who now were living near Leavenworth, refused to move to the Council Bluffs reservation. Potawatomi that were scheduled to be relocated to the Platte Country but were still living around the St. Joseph and Wabash Rivers also refused the new destination. Under the direction of a Baptist missionary, the Reverend Isaac McCoy, these groups agreed to settle in a newly created reservation in eastern Kansas, adjacent to the Osage River (Clifton 1998).

The Council Bluffs region in the early 1840s was hundreds of miles from any white settlements, missions, or government officials, which suited the United Band, who desired greater autonomy from the American government and the freedom to maintain their traditional culture. At Council Bluffs, outside contact with the rest of the country was lacking for most of the year; even the Indian agents only spent a part of the year there. In their desire to isolate themselves culturally from all outsiders, the United Bands also refused to cooperate with





possibility of hostilities as justification for yet another removal, and plans to relocate the United Bands were being developed by 1839 (Mitchell 1995). Thus almost as soon as the United Bands arrived in Iowa, there was federal pressure for them to move once more, to the National Reserve on the Osage River in Kansas. The threat of warfare with the Dakota, combined with the proliferation of farming settlements in Iowa and the push for Iowa statehood, prompted government officials to compel these Potawatomi to move again. The desire to get the



**FIGURE 2-8. Gunn and Mitchell's map showing the Potawatomi National Reserve in Kansas. (Courtesy Wichita State University Libraries)**



Potawatomi out of Iowa was strong enough to allow the United Bands to negotiate from a position of power, and they were able to negotiate a purchase price for their land that was more than three times the original government offer (Clifton 1998; Mitchell 1995).

In 1846 a treaty was signed by two separate Potawatomi bands relinquishing both the Council Bluffs and Osage River reserves. A new reservation for both groups was to be located along the Kansas River in northeast Kansas (Figure 2-8). A number of Potawatomi in the Great Lakes region had never made the migrations west, primarily due to the transient ownership of the new lands. The last official relocation of Potawatomi from Wisconsin did not occur until 1852, to the new Potawatomi National Reservation along the Kansas River. Potawatomi from the Council Bluffs area settled in the northeast corner of the reserve, along tributaries of Soldier Creek north of the Kansas River. Potawatomi from the Osage River reserve settled the lands south of the Kansas River (Clifton 1998; Mitchell 1995). Whereas the United Bands from Council Bluffs wished to retain their traditional culture, and were wary of non-Indian institutions and influence, the Potawatomi from the Osage River reserve had been heavily influenced by missionaries and were more accepting of white culture. Due to the cultural differences that had arisen over time from these opposed traditions, the United Band argued that they could not live with their relatives from Indiana, known as the Mission Band Potawatomi (Manzo 1981; Mitchell 1995).

Ironically, preparation of new Indian lands by U.S. officials made the government aware of the value of these lands. The concept of the plains as a Great American Desert was soon abandoned, and in these Indian lands Americans began to see opportunity. The eastern Indians who had resettled in Kansas soon found American settlers clamoring for their lands. Due to their legal land reserves, these immigrant Indians were seen as more problematic to white settlement

than the nomadic Indians of the Plains. New treaties were negotiated in 1853 and 1854 that led to the cession of 13 million acres of land belonging to the Delaware, Iowa, Kickapoo, Odawa, Shawnee, Wyandotte, Sac and Fox, and other tribes formerly from the east (Clifton 1998; Miner and Unrau 1978).

The Kansas-Nebraska Act of 1854 opened the region to American settlement. The political climate of the time encouraged settlement in Kansas primarily for popular sovereignty relating to slavery, quickly increasing the pressure on remaining Indian groups to sell their recently acquired lands (Fixico 2003). Squatters appeared on Potawatomi reserve lands near Topeka, which had been founded as a town association in 1854. Among the founders of the town was Cyrus K. Holliday, a future mayor of Topeka and founder of the Atchison, Topeka & Santa Fe Railroad. As the white population in the area grew, Indian agents claimed to have little authority to prevent the squatters (Miner and Unrau 1978; Snell and Wilson 1968). The Potawatomi expected another assault on their lands and increasing pressure on them to move further west. Railroad companies were also vying for Indian lands. The railroads desired rights-of-way through Indian territories to connect the eastern and western sections of the United States. Railroads also attracted white laborers, as well as settlers who would be able to utilize the trains to transport their agricultural products to distant markets (Mitchell 1995).

The Potawatomi of the United Bands had been more successful in avoiding U.S. governmental efforts to assimilate them. Government officials saw them as an unmanageable lot always asserting their independence and rejecting American attempts at assimilation. These Prairie Band Potawatomi were considered to be a bad influence on the missionized Potawatomi to the south. In particular, the Prairie Band rejected farming, and denigrated those who adopted this form of subsistence (Clifton 1998; Connelley 1918). The new term “Prairie Band” likely

arose from the name “Prairie Indians of Caldwell’s Band of Potawatomies,” a name the tribe petitioned the commissioner of Indian affairs to give them following the death of revered half-breed Potawatomi leader Billy Caldwell (Sauganash) in 1841 (Clifton 1998).

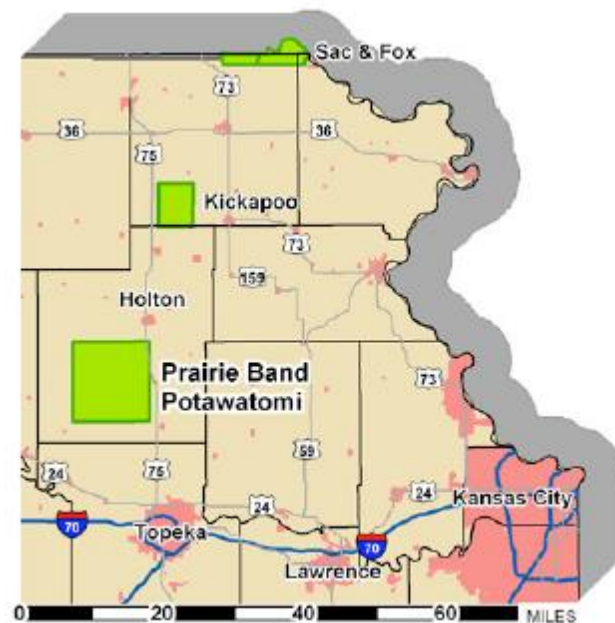
Despite reuniting on the National Reserve, cultural and religious differences had developed between the United Bands and the Mission Band that eventually led to a permanent split in the tribe. Differences between the two groups were as obvious as their footwear: the conservative United Band members preferred moccasins; the Mission Band in contrast wore American-made shoes. By 1861, 1,400 of the 2,170 Potawatomi living on the National Reserve had decided to accept individual land allotments and to become citizens of the U.S. These individuals, primarily of the Mission Band, would thus become known as the Citizen Potawatomi. The remaining 780 preferred to continue to hold their land in common and rejected any offer of U.S. citizenship. Primarily from Council Bluffs, these Potawatomi looked at the effects of past treaties and concluded that surrendering land in another treaty would be a mistake (Connelley 1918; Mitchel 1995).

Nonetheless, in 1861—the same year Kansas was granted statehood—the Potawatomi signed a treaty reducing their National Reserve. The Prairie Potawatomi fought to keep their section of land in common, but the Mission Potawatomi chose to take 152,128 of the 498,642 acres as individual allotments, to dispose of as they saw fit. The Prairie Band kept 77,358 acres, and the remaining 269,156 acres were distributed to corrupt government agents and finally sold to a railroad company. The Jesuits also received over 2,000 acres of the unallotted lands. From this point on the Prairie Band Potawatomi were officially and legally a separate entity from other Potawatomi groups (Clifton 1998; Mitchell 1995).

At the 1861 treaty negotiations, the Indian commissioner had hoped to convince the Prairie Band to agree to the allotment of their lands and accept U.S. citizenship as the Mission Potawatomi had done. He suggested that the Prairie Band sell the remaining lands of their Kansas reservation and purchase new lands farther to the west, but the Prairie Band leadership rejected both options, determined to hold on to their land in common. They were able to negotiate this concession in the 1861 treaty, keeping in common 11 square miles in the northeast corner of the National Reserve (Figure 2-9). The Prairie Band would sign no further treaties with the U.S. (Clifton 1998; Connelley 1918).

### **Fighting to Maintain Tribal Integrity**

The 1860s saw a major reduction in the Indian population in Kansas. The Indian Removal from the East during the 1830s was repeated in Kansas during subsequent decades.



**FIGURE 2-9. The reduced Prairie Band Potawatomi Reservation in northeastern Kansas. (Courtesy Prairie Band Potawatomi Nation Division of Planning and Environmental Protection)**

Prior to the Civil War Kansas was home to about 10,000 Indians. By 1875 there were fewer than 1,000 (Miner and Unrau 1978). In 1865 780 Prairie Band lived on the reservation, but their population was reduced to 400 at the beginning of the 20th century, primarily as a result of out-migration (Clifton 1998).

After the Civil War, members of Congress began to argue that the Indians no longer displayed the attributes of sovereignty and that treaties should no longer be made with the tribes. Subsequently, the treaty era ended in 1871 with an amendment to the Indian Appropriations Act. This act of Congress specified that tribes would no longer be treated as independent nations with which the U.S. government could negotiate treaties. All subsequent Indian policies would be negotiated through congressional legislation, thereby removing Indians from the negotiation process: after 1871 the federal government dictated the terms of Indian policy to the tribes through congressional acts and presidential orders (Clifton 1998; Prucha 1997; Stull 1984). Yet despite an 1872 statement by Secretary of the Interior Columbus Delano that their tribe had become extinct, the Potawatomi tenaciously hung on to their Kansas lands and tribal structure (Miner and Unrau 1978).

The Dawes Severalty Act, or General Allotment Act (24 Stat. 388) of 1887, authorized the survey of tribal lands for partitioning into individual plots in an attempt to establish Indians as land-owning farmers. The U.S. secretary of the interior was obliged to purchase any remaining Indian lands not allotted to individuals. The surplus lands were sold to settlers, and the funds from these sales were to be held in trust for the purpose of providing money for Indian welfare, education, and the process of civilization (Fiorentino 1999; Meriam et al. 1928; Tyler 1973; Washburn 1986). The Dawes Act also encouraged Indian people to become U.S. citizens. Prairie Band leaders vehemently opposed it and attempted to thwart its implementation on the

reservation (Clifton 1998). The Prairie Band leader Wahquahboshkuk fought against the breakup of the tribe. He resisted the white institutions of education and Christianity and worked against those who supported allotment (Herring 1990).

Regardless, in 1890, against the wishes of the Prairie Band, the reservation was ordered allotted, and any remaining acreage was to be sold. Over 800 such allotments had been made by 1904. Those who opposed allotment often ignored the orders, but the Indian agent filed the paperwork in the names of the allottees regardless. Of the allotments made, nearly half of the first 100 were sold to outsiders, effectively taking the land out of the hands of the Prairie Band. By the early 20th century the allotment process was considered complete, and the U.S. government no longer recognized the Prairie Band Potawatomi as a tribe (Clifton 1998; Connelley 1918).

Legislation such as the Dawes Act indicated that Indian culture was to be completely replaced by white culture. Not only was a new subsistence method to be forced upon the Native Americans, they were expected to adopt the social organization, religion, behavior, and attitudes of the dominant U.S. society (Prucha 1976). Euro-American notions of morality, responsibility, industry, child rearing, and gender roles were different from those of the Potawatomi, however, and the Prairie Band, in particular, were not willing to give up their culture so readily. Many continued their old subsistence practices as best they could and also practiced traditional religious and social customs in private. Adoption of white farming practices was done as much for appearance as for subsistence. After generations of learning that openly resisting U.S. government policies was futile, the Prairie Band realized they could maintain their cultural identity if they acquiesced just enough to keep the whites satisfied. This was an adaptive strategy that would allow the Prairie Band to maintain aspects of their heritage and identity

throughout the 20<sup>th</sup> century, when other Kansas Indian tribes were being relocated or disbanded (Clifton 1998; Mitchell 1995).

The Dawes Act was intended to break up the tribal system and to establish individual Indians as independent farmers and productive U.S. citizens, but it ultimately resulted in land deprivation and poverty. Instead of becoming integrated into the greater American culture, the Prairie Band were forced to cede its tribal self-determination to the U.S. government, which became the dictator of its affairs (Clifton 1998).

Some Prairie Band took up farming after the departure of the Citizen Potawatomi, but few had received training in how to farm. With little experience or knowledge of farming, many Potawatomi chose to sell or lease their lands to white farmers who were generally able to procure Prairie Band lands at prices well below their actual value. Thus the Potawatomi often had little money to show from leasing or selling their land. Farming and land ownership brought few economic gains to the Prairie Band. Most lived in extreme poverty and felt helpless to improve their lot (Mitchell 1995). The sale of allotted lands to whites accelerated following the turn of the century (Connelley 1918), especially after Prairie Band men returned to the reservation following military service during World War I. The returning soldiers found few economic opportunities on the reservation, sold their land for cash, and moved to cities where more work was available. Employment opportunities in cities lured a number of Prairie Band away from the reservation and into the wider American society (Mitchell 1995).

The Great Depression was a difficult time for the Prairie Band, as it was for much of the U.S. population. However the Prairie Band, already used to poverty and having no money to lose in bank failures, were able to survive the crisis as they had survived many previous assaults on their economic well-being. Many hunted wild animals and sold skins for money, while others

developed gardens that provided food as well as a small surplus, which could be sold (Mitchell 1995). The Dust Bowl droughts of the 1930s also brought hardship to the Prairie Band Reservation. When confronted with repeated droughts and grasshopper infestations, family farming operations that were already marginal failed completely. Few farms based on intensive agriculture or ranching survived these times. Many tribal members survived by subsistence farming, planting a wide variety of crops as well as keeping chickens and milk cows. Hunting wild game also contributed to subsistence. Families with children could save money by sending them to Indian boarding schools, thus transferring the costs of their food and clothing to the Bureau of Indian Affairs, although boarding schools adversely affected the culture of Indian people by suppressing Indian culture and language among the children (Mitchell 1995).

John Collier was named commissioner of Indian Affairs by President Franklin D. Roosevelt in 1933 (Tyler 1973). President Roosevelt's 1934 New Deal to provide relief to Americans suffering from the Great Depression came with a New Deal for Indian people: the Indian Reorganization Act (IRA). The primary employment on the Prairie Band Reservation at the time would come from New Deal programs such as the Works Progress Administration (WPA) and Civilian Conservation Corp (CCC). The Potawatomi Community Building was one such WPA project (Mitchell 1995). Despite the fact that the Indian Reorganization Act provided many of the rights that Prairie Band *okamek* such as Wakwaboshkok had fought for several decades earlier, the conservative leaders of the tribe in the 1930s rejected the new Indian policy (Clifton 1998). They opposed the Indian Reorganization Act because the manner in which the tribal governments were to operate ran counter to traditional Potawatomi views of governing. Throughout most of Potawatomi history, political decisions were made by achieving a consensus among all adult tribal members. The concepts of leadership councils and the democratic vote



were foreign to the Prairie Band; older, more conservative members of the tribe were suspicious of this new form of governance, which was to be imposed from the outside. Additionally, the traditional Prairie Band did not see the Indian Reorganization Act as establishing Indian self-determination. To them, independence could not be achieved as long as the Bureau of Indian Affairs dictated their system of governance and oversaw all tribal decisions (Mitchell 1995).

Moreover, these traditional leaders had already developed their own governmental body as described in the Baldwin Constitution (named for Haskell Indian Institute superintendent R.D. Baldwin, who helped draw up the document). This 1932 compact organized a tribal advisory committee to be elected by adult members of the tribe. The major motivation for the tribal committee was to legitimize those tribal members who wished to pursue legal action against the federal government. The members of the newly created tribal advisory committee were attempting to control the resources that would come from a new source: litigation. However, a major flaw with the short-lived Baldwin Constitution was that it failed to specifically define tribal membership, setting the stage for less conservative, off-reservation members to assume positions of power (Clifton 1998). Controversy over the election of new advisory committee members led the superintendent to remove the incumbents from their positions; thus during World War II the tribal advisory committee ceased to operate.

Nonetheless the Prairie Band were quick to pursue litigation against the U.S. government for past injustices. Former members of the tribal advisory committee and their families attempted to resurrect the committee to pursue litigation and the anticipated assets from lawsuits. After the conclusion of World War II, many Prairie Band returned to the reservation, among them younger, more progressive tribal members; a power struggle subsequently ensued with the conservative members of the tribal advisory committee. The progressive elements of the tribe

asserted that the tribal advisory committee did not represent the majority of Prairie Band members and did not adhere to the Baldwin Constitution that supposedly legitimized their power (Clifton 1998; Mitchell 1995).

Following the resignation of John Collier in 1945, Congress and the executive branch renewed their efforts to assimilate Indians into American society. The new policy was a direct rebuttal of Collier's New Deal policies, which many in government viewed as aberrant (Prucha 1984). The Commission on Organization of the Executive Branch of the Government, otherwise known as the Hoover Commission, examined Indian affairs as a part of their analysis of federal departments. The Hoover Commission viewed assimilation as a primary goal for alleviating Indian economic and health disparities and established the modern concept of termination to that end (Deloria 1985; Tyler 1973). Termination policy was aimed at assimilating Indians into American society by completely terminating the federal trust relationship with tribes, eliminating reservations, and encouraging Indian people to move into urban areas. Proponents of termination argued that eliminating reservations and encouraging Indians to seek work in cities would hasten assimilation and lead to the abandonment of the traditional tribal culture. Indian adoption of the economic, social, and cultural systems of the greater U.S. populace would thus free the government from administrative and economic oversight of Indian affairs (IHS 2005).

Among the first moves the federal government made toward termination was to establish the Indian Claims Commission, created in 1946 by H.R. 4497. The Indian Claims Commission Act allowed Indian tribes to bring legal claims against the U.S. government, but it was also an attempt by the government to end its involvement in Indian affairs (Clifton 1998; Mitchell 1995). The Meriam Report of 1928, a survey of problems in Indian administration, had called for reform of the protracted and unwieldy process for adjudicating Indian legal claims against the

government. Established in 1946, the Indian Claims Commission Act created a three-man commission to hear and evaluate Indian legal claims (Prucha 1997; Tyler 1973). Most of the lawsuits concerned the inadequacy of compensation to the Indians for ceded or seized lands. Though the Indian Claims Commission allowed the tribes to sue for past injustices, it only paid out the value of lands at the time they were seized (Stull 1984; Tyler 1973). Some Congressmen argued that the promise of payment of claims led to idleness on the part of the Indians, and delays in the hearing of claims prolonged their indolence. Others viewed the Indian Claims Commission as a necessary part of preparing the tribes to become less dependent on the federal government (Prucha 1997; Tyler 1973). In fact, the settlement of Indian claims would allow the government to terminate the special status of Indians as federal wards, transforming them into equal citizens of the U.S., and freeing the government from any further obligations (Kane and Kane 1972).

House Concurrent Resolution (HCR) 108 was the primary legislation of the termination policy. It stated explicitly that Indians would be subject to the same laws, rights, and responsibilities as all other citizens of the U.S., and that their special status as wards of the U.S. government should end. Tribes would cease to be legal entities, as far as the government was concerned. By the 1950s the mission of the Bureau of Indian Affairs had become the removal of Indian people from reservations, their integration into the greater U.S. society, and termination of federal management of Indian affairs (Clifton 1998).

As early as 1940 the federal government conferred upon the state of Kansas concurrent jurisdiction over crimes committed on Indian lands (18 U.S.C. 3243). Such laws foreshadowed the termination legislation to come (Stull 1984). Starting in 1947 the Prairie Band became the target of legal termination by the federal government. In 1954, HR 4985 was adopted to

terminate five specific tribes. Named in the House resolution were the Potawatomi of Kansas. Conservative leaders of the Prairie Band such as Minnie Evans fought against termination and argued to maintain the tribal structure. These efforts brought a rare success for the Prairie Band, preventing the completion of their termination (Mitchell 1995). Despite these efforts, allotment left only 22% of the Prairie Band reservation under Indian control in 1960 (Stull 1984).

Freeing Indians to pursue the American Dream under the auspices of termination actually deprived Indian people of the funds, lands, and services promised to them by the federal government (Prucha 2000; Stull 1984). The termination policy resulted in the sale of more tribal lands, because state governments assumed legal authority over and the right to tax the terminated lands. The policy led to the devastation of tribal communities, customs, languages, and cultures through impoverishment and denial of Indian tribes as legal entities and as distinct cultural groups. During the termination period, the U.S. Congress passed legislation that terminated the federal trust relationship for 109 tribes (IHS 2005).

## **Self-Determination**

Not until 1961 were the progressive elements in the tribe able to secure a new constitution. The new constitution defined tribal members as being anyone who had received allotments in accordance with the Dawes Act and their descendants. Degree of Indian blood was not a requirement. Interestingly, many of those who came to positions of power after the 1961 constitution were descended from the people who accepted the Dawes Act and rejected the attempts of leaders such as Wakwaboshkok to maintain the tribal system. The 1961 constitution was an opportunity for the Prairie Band to assert control over its own fate, and perhaps improve cultural and economic living conditions. By 1975 the Prairie Band had yet to prosper from the

new government and had accomplished little socioeconomically. Internal dissent and resistance to outsiders and change persisted during this period (Clifton 1998).

The Office of Economic Opportunity's (OEO) Indian Division started working in Kansas in 1965. This organization began promoting government-funded programs designed to improve the economic status, health, education, and living conditions among the Potawatomi, but the Prairie Band viewed the OEO as another version of the federal Indian agent. Many Prairie Band members worked to thwart and sabotage OEO efforts in an attempt to drive the organization away. Thus the OEO resources had little effect on the conditions of the Prairie Band reservation (Clifton 1998).

After 1962 the Prairie Band received a \$4.1 million settlement from the 1829 Prairie du Chien treaty [(11 Ind. Cl. Comm. 693, 710 (1962))]. In 1967 each Prairie Band member began to receive per capita payments from the interest from a claims settlement over the 1846 treaty totaling \$1,176,788. The 2,101 members would each receive \$490.50 (Clifton 1998).

In 1970 the Nixon administration introduced a program of self-determination for Indian people. Prairie Band leaders thought this might usher in a new era of change. But working within the system had been very frustrating for many in the tribe, and they turned against what they saw as the inept leadership of the Bureau of Indian Affairs and their own tribal government. A dispute with the bureau over Jesuit lands that had been returned to the tribe led to political chaos on the reservation (Mitchell 1995). In the early 1970s some conservative members began to follow the example set by the American Indian Movement (AIM), forming the Tribal Action Committee. In the summer of 1972 the committee occupied the Indian Office in Horton, Kansas. Shortly thereafter, the commissioner of Indian affairs suspended the 1961 constitution as the conservative and more militant faction of Prairie Band fought against the progressive and

absentee groups for control of Prairie Band governance (Clifton 1998). With the constitution suspended, the BIA ceased providing money for federal programs to the Prairie Band, and poverty persisted (Mitchell 1995). Not until 1977 did the BIA approve a new tribal constitution. Shortly thereafter, the tribe was able to take advantage of congressional legislation that included the Indian Self-Determination Act (1975), the Indian Health Care Improvement Act (1976), the Indian Child Welfare Act (1978) and the Indian Religious Act (1978). Federal money again began to trickle into the reservation, and reforms began (Mitchell 1995). After 1980, however, the Reagan administration reduced the budget of the BIA, reducing the funds available on the Prairie Band Reservation.

The unreliable nature of government funds prompted the Prairie Band to seek alternative funding. Outside companies had operated bingo ventures on the reservation with little success. In 1987 the tribe decided to take over the bingo enterprise and run it themselves. Bingo provided the Prairie Band with a relatively stable source of income, and thus began an era of economic growth for the tribe (Mitchell 1995). The Indian Gaming Regulatory Act of 1988, legalized the operation of gambling casinos by Indian tribes as a way to promote economic development and self-sufficiency. By 1992 the Prairie Band had worked out a contract with the state of Kansas to allow a gambling venture on the reservation, but legal challenges postponed legislative approval of the contract until 1995 (Mitchel 1995). Soon thereafter the Prairie Band entered into a deal with Harrah's Entertainment, Inc. to build a casino on the reservation. Harrah's Prairie Band Casino opened in 1998.

The casino provided the tribe with a new source of revenue: 43% of revenue is dedicated to economic infrastructure development and 30% goes to per capita payments to individual tribal members (Heck 2007). There are currently almost 5,000 enrolled members of the Prairie Band

Potawatomi Nation. Enrollment declined considerably following a May 2000 amendment to the constitution, which made it necessary for members to possess at least 1/4 PBP Nation blood (Prairie Band Potawatomi Nation 2011).

Despite the more stringent tribal membership rule, the community on and around the reservation includes many Native Americans from other tribes. In particular, the Prairie Band have had a long and close relationship with the Kansas Kickapoo (Clifton 1969). By the mid-1960s, well before the changes in enrollment, “exclusive” Prairie Band households only constituted 30% of the households on the reservation. Prairie Band and Citizen Potawatomi households constituted 19%; and Prairie Band and Kickapoo households made up an additional 11%. Households consisting of Prairie Band and members of other (non-Kickapoo) tribes were 19% of the reservation total. The remaining 21% consisted of Prairie Band members living with non-Natives (Clifton and Isaac 1964). Although Native non-members of the Prairie Band tribe cannot receive PBP per capita payments, they can receive BIA, Indian Health Service, and other federally funded services provided on the reservation.

## Chapter 2 References Cited

Berry, Mary F., Reynoso, C., Braceras, J.C., Edley, Jr., Kirsanow, P.N., Meeks, E.M., Redenbaugh, R.G., and Thernstrom, A. 2004. *Broken Promises: Evaluating the Native American Health Care System*. U.S. Commission on Civil Rights. Washington, D.C.: U.S. Government Printing Office.

Calloway, Colin G. 1995. *The American Revolution in Indian Country: Crisis and Diversity in Native American Communities*. New York: Cambridge University Press.

Clifton, James A. 1969. Sociocultural Dynamics of the Prairie Potawatomi Drum Cult. *Plains Anthropologist* 14(44):85-93.

-- -- --. 1998. *The Prairie People: Continuity and Change in Potawatomi Indian Culture, 1665 - 1965*. Iowa City: University of Iowa Press.

Clifton, J.A. and Isaac, B. 1964. The Kansas Prairie Potawatomi: On the Nature of a Contemporary Indian Community. *Transactions of the Kansas Academy of Science* 67(1):1-24.

- Connelley, W.E. 1918. The Prairie Band of Pottawatomie Indians. *Collections of the Kansas State Historical Society 1915-1918* 14:488-570.
- Deloria, Vine 1985. "The Evolution of Indian Policy Making," in *American Indian Policy in the Twentieth Century*. Edited by Vine Deloria, pp. 239-256. Norman: University of Oklahoma Press.
- Edmunds, R.D. 1978. *The Potawatomis: Keepers of the Fire*. Norman, OK: University of Oklahoma Press.
- Fiorentino, Daniele 1999. *Acculturation/Assimilation: American Indian Policy in the Progressive Years*. Ph.D. dissertation, Department of History, University of Kansas.
- Fixico, D.L. 2003. American Indians in Kansas. *Kansas History* 26(4):272-287.
- Goddard, I. 1972. Historical and Philological Evidence Regarding the Identification of the Mascouten. *Ethnohistory* 19: 123-134.
- Heck, S. 2007. *PBP Directors of Departments and Programs Bracing for 2008 Budget Cuts*. Prairie Band Potawatomi News, November 2007 Edition, 1.
- Herring, J.B. 1990. *The Enduring Indians of Kansas: A Century and a Half of Acculturation*. Lawrence, KS: University Press of Kansas.
- Indian Health Service (IHS) 2005. *The First 50 Years of the Indian Health Service: Caring and Curing*. Rockville, Md.: U.S. Department of Health and Human Services.
- Kane, Robert L., and Kane, Rosalie A. 1972. *Federal Health Care (With Reservations!)*. New York: Springer Publishing Company.
- Manzo, J. T. 1981. Emigrant Indian Objections to Kansas Residence. *Kansas History* 4(4):246-254.
- Meriam, L., Brown, R.A., Roe Cloud, H., Dale, E.E., Duke, E., Edwards, H.R., McKenzie, F.A., Mark, M.L., Ryan Jr., W.C., and Spillman, W.J. 1928. *The Problem of Indian Administration*. Report of a Survey at the Request of Honorable Hubert Work, Secretary of the Interior, and Submitted to Him, February 21, 1928. Washington, D.C.: Institute for Government Research.
- Miner, H.C. and Unrau, W.E. 1978. *The End of Indian Kansas: A Study of Cultural Revolution, 1854 – 1871*. Lawrence, KS: The Regents Press of Kansas.
- Mitchell, G.E. 1995. Stories of the Potawatomi People: From Early Days to Modern Times. Originally published in the Topeka Capital Journal. <http://www.kansasheritage.org/pbp/books/mitch/mitchbuk.html>
- Prairie Band Potawatomi Nation. 2011. Tribal Administration. Edited by S. Heck. November 29, 2011 <http://www.pbpindiantribe.com/tribal-administration.aspx>
- Prucha, Francis P. 1976. *American Indian Policy in Crisis: Christian Reformers and the Indian, 1865 – 1900*. Norman, OK: University of Oklahoma Press.
- . 1984. *The Great Father: The United States Government and the American Indians*, Vols. 1 and 2. Lincoln: University of Nebraska Press.



- . 1997. *American Indian Treaties: The History of a Political Anomaly*. Berkeley: University of California Press.
- . 2000. *Documents of United States Indian Policy*, 3rd edition. Lincoln: University of Nebraska Press.
- Snell, J.W. and Wilson, D.W. 1969. The Birth of the Atchison, Topeka, and Santa Fe Railroad. *Kansas Historical Quarterly* 34(2):113-142.
- Stull, D.D. 1984. *Kiikaapoa: The Kansas Kickapoo*. Horton, KS: Kickapoo Tribal Press.
- Tyler, S. Lyman 1973. *A History of Indian Policy*. Washington, D.C.: Bureau of Indian Affairs, United States Department of the Interior, U.S. Government Printing Office.
- Washburn, Wilcomb, E. 1986. *The Assault on Indian Tribalism: The General Allotment Law (Dawes Act) of 1887*. Malabar, Fla.: Robert E. Krieger Publishing Company.

## **Chapter 3: Epidemiologic Transition**

This chapter presents epidemiologic transition theory as it will be applied to the data for the Prairie Band Potawatomi community in subsequent chapters. Epidemiologic transition theory attempts to explain changes in the primary causes of death in a population over time. The level of analysis is the population. That is, the theory describes the kinds of diseases that are the primary causes of death in a population at a given point in time. The theory also describes how the causes of mortality change over time, and attempts to explain when and why the changes occurred. Epidemiologic transition is a process thought to be closely related to human subsistence patterns and social arrangements. The Theory of Epidemiologic Transition was first put forth by Abdel R. Omran (1971), but previous years of research and discoveries led up to the delineation of the theory.

Epidemiology investigates how diseases are distributed in populations, the causes and costs (financial and in human suffering) of diseases, and how diseases can be prevented or reduced. Its focus includes all of the related factors of health and disease. Epidemiology is also concerned with fluctuations in health and disease that occur within populations over time. An epidemiologic transition occurs when infectious diseases such as typhoid, tuberculosis, cholera, diphtheria, and plague decline as the leading causes of morbidity and mortality and are replaced by heart disease, cancer, stroke, diabetes, gastric ulcer, and other chronic non-infectious diseases. Also more common after the transition are mental illness, accidents, addiction, and diseases due to industrial exposure or a deteriorating environment (Omran 1977b).

The theory of epidemiologic transition grew out of years of research and a scientific approach to understanding and explaining disease causation. Omran (1971) expanded and elaborated upon demographic transition theory as first proposed by Thompson (1929) and later

Notestein (1945). Omran (1971) first coined the term “Epidemiologic Transition” and was the first to describe its characteristics. His model integrated epidemiology with demographic changes among human populations (Barret et al. 1998). He suggested that part of the process of modernity is a change in the types of diseases that cause the preponderance of deaths in a society (Broudy and May 1983) and set forth five propositions regarding the epidemiologic transition, each based on known facts regarding the relationship between disease and population dynamics:

*Proposition One:* Mortality is a fundamental component of population makeup.

Oscillations in mortality first become less frequent and less dramatic. The initial decline in mortality, at first slow and barely noticeable, gradually accelerates. Mortality eventually levels off at low frequencies in the twentieth century.

*Proposition Two:* A long-term change occurs in disease and mortality patterns where chronic degenerative diseases slowly replace infectious disease outbreaks as the primary cause of death. Omran (1971) described three ages of mortality patterns:

1. *Age of Pestilence and Famine:* Mortality is high and variable, thus preventing sustained increases in population size. Average life expectancy at birth is low and variable, ranging between 20 and 40 years.
2. *Age of Receding Pandemics:* Mortality drops continually and the rate of decline speeds as epidemics become less common or disappear altogether. Average life expectancy at birth steadily increases from 30 to 50 years. Population grows exponentially.
3. *Age of Degenerative and Man-Made Diseases:* Mortality declines and eventually becomes stable at a low level. Average life expectancy at birth increases slowly until it

exceeds 50 years. During this age fertility becomes the principal cause of population increase.

*Proposition Three:* The most dramatic changes in disease patterns are among young women and children.

*Proposition Four:* Shifts in morbidity and mortality are closely interrelated with the demographic and socioeconomic transitions that are a part of the process of modernization.

*Proposition Five:* Variations in the pattern, speed, determinants and consequences of population change among different populations delineate three models of the epidemiologic transition:

1.     The Classical (Western) Model of Epidemiologic Transition: The Classical model is characterized by a gradual, progressive shift from high mortality (above 30 per 1000 people/yr) and high fertility (above 40 per 1000/yr) to low mortality (less than 10 per 1000/yr) and low fertility (less than 20 per 1000/yr). The shift occurred in conjunction with the process of modernization in most Western societies.
2.     The Accelerated Epidemiologic Transition Model: A major component distinguishing the accelerated model is that the time required for mortality to drop to 10 per 1000 people is much less than that for the classical model. The accelerated model is most often associated with the mortality transition as it occurred in Japan.
3.     The Contemporary (or Delayed) Epidemiologic Transition Model: The Delayed model is characterized by the rapid, significant decreases in mortality that have been observed among Global South nations since the end of World War II.

Omran (1971) identified three categories of disease determinants:

1. Ecobiological determinants of mortality include the complex interactions between infective disease agents, the environment, and the resistance of the host species.
2. Socioeconomic, political, and cultural determinants include such things as standard of living, health behaviors, hygienic practices, and nutritional intake.
3. Medical and public health determinants include specific preventive measures and treatments used to fight disease. They include improved sanitation, immunizations, and the development of curative therapies.

Mortality reduction in Europe and other Western nations during the 19th century, as seen in the classic model of epidemiologic transition, was the result primarily of ecobiologic and socioeconomic determinants (Omran 1971). The decline of plague and other pandemics in Europe was most likely not the result of medical breakthroughs, according to McKeown and Brown (1955). The decline of mortality in the Global South has occurred more recently and is primarily the result of medical interventions: the effects of biomedicine have been more direct and significant and are typical of the contemporary or delayed transition model (Omran 1971).

Omran also introduced a “transitional variant of the delayed model” in 1983. This model described the transition among such nations as China, Taiwan, Korea, Singapore, Hong Kong, Mauritius, Sri Lanka, and Jamaica. Mortality declined rapidly in these countries after 1940, typical of the contemporary or delayed model. However in this transitional variant of the delayed model a fertility decline occurred in these nations just a few decades after the mortality decline. Social development and organized family planning efforts very likely played important

roles in the fertility decline. Nor did mortality slow in the same manner in these nations as it did in other nations exhibiting the delayed model (Omran 1983).

## **The Age of Pestilence and Famine**

With the shift to agriculture 10,000 years ago came the *Age of Pestilence and Famine*. Mortality was primarily the result of epidemics, wars, and famines (Omran 1971). This was Omran's first Age, and is generally referred to as the first epidemiologic transition, due to a dramatic shift in disease prevalence never before experienced by modern human populations (Armelagos 2004; Armelagos, Barnes, and Lin 1996; Barret et al. 1998). Most of the infectious diseases that have plagued humankind throughout history became prominent during this period (Omran 1977b).

Cholera, croup, diarrhea, dysentery, erysipelas, influenza, intermittent fever or ague (malaria), measles, scarlatina, smallpox, syphilis, thrush, typhoid, whooping cough, yellow fever, mumps and other forms of parotitis were thought to be of zymotic or miasmatic origin. Consumption (tuberculosis), bronchitis, and pneumonia were considered to be respiratory diseases. U.S. census data from 1850 for several large cities and the states indicate that these two categories of disease were the cause of approximately half of all deaths at that time (Bettmann 1974; Omran 1977b). Heart disease of the arteriosclerotic variety familiar today was rare during this age. Heart conditions were more likely due to rheumatic fever following a streptococcal infection, often affected children and could damage the heart valves (Omran 1971).

Tuberculosis generally was the leading cause of death, particularly among adolescents and young adults, pregnant women, minorities, and the poor. Tuberculosis was especially virulent in young females in their fertile years. It certainly took a greater toll among young females than among males and was justifiably called the "Captain of the Men of Death" and the

“white plague,” which referred to the view of the disease as being a sort of divine suffering (Bettmann 1974; Omran 1971, 1975, 1977b). Only after the beginning of the 20th century was tuberculosis recognized as an infectious disease (Condran and Cheney 1982). Even into the 20th century, many continued to view tuberculosis as a constitutional disease with a significant genetic component (Ewbank and Preston 1990).

Diseases due to nutritional deficiencies were common (Omran 1971). But a characteristic peculiar to the early transition in America was the near absence of famine, which was typical of this stage of the transition in Europe. The absence was likely due to the greater availability of food and agricultural products for the comparatively smaller U.S. population (Omran 1977b).

## **The Age of Receding Pandemics**

The Industrial Revolution began in the late 18th century in England and spread to Europe and North America during the 19th century. The *Age of Receding Pandemics* also occurred at about this time, with industrializing nations experienced a significant decline in deaths due to infectious diseases (Barret et al. 1998). Norway was the first European nation to show this shift (Pitkanen 2002). Thus began the decline in infectious disease mortality and the increase in chronic disease mortality that would produce the second epidemiological transition.

European mortality due to infectious diseases declined over three phases starting late in the 17th century. The first phase lasted until the beginning of the 19th century and is typified by reductions in mortality due to intermittent disease epidemics. The second phase began in the mid-19th century and was distinguished by a steady secular mortality decline as the population continued to modernize. With the development of antimicrobial drugs in the 1940s, the third phase of mortality decline began (Schofield and Reher 1991). The *Age of Receding Pandemics*

began later in the U.S. than in Europe, likely during the middle of the 19th century. Mortality began to decline at this time and the pattern continued until 1920 (Omran 1977a, 1977b).

During the early phase of the *Age of Receding Pandemics*, mortality from tuberculosis reached its peak as nations industrialized. Tuberculosis at this time was still most deadly to young women. Heart disease remained infrequent, and there was a high rheumatic to arteriosclerotic ratio. Starvation was less frequent than before but nutritional-deficiency diseases were frequent (Omran 1971). During the later phase of the *Age of Receding Pandemics*, diseases such as cholera and yellow fever vanished, and scarlet fever became less virulent. The incidence of malaria, typhoid, and typhus declined and many childhood diseases were reduced. Except during the 1918 – 1919 influenza pandemic, tuberculosis mortality declined, but was still highest in young women. Heart disease mortality increased, and the rheumatic to arteriosclerotic ratio decreased. Death due to starvation became rare, and diseases due to nutritional deficiencies began to fade (Omran 1971; 1977).

McKeown (1976, 1979) examined mortality change in England and Wales during the 19th century. He calculated the percentage of the decline in standardized mortality rates by cause, and found that 75% of the mortality decline was the result of decreases in infectious disease deaths. Other nations experienced similar declines, albeit not all at the same time. Over the course of 200 years and across nations, two-thirds of the decline in mortality could be attributed to decreases in infectious and parasitic illnesses (Puranen 1991). Tuberculosis mortality declined dramatically during this stage and was the most significant decline of all infectious diseases (Puranen 1991). Condran and Cheney (1982) found that the decline in tuberculosis mortality was responsible for 26.8% of the total mortality decline in Philadelphia



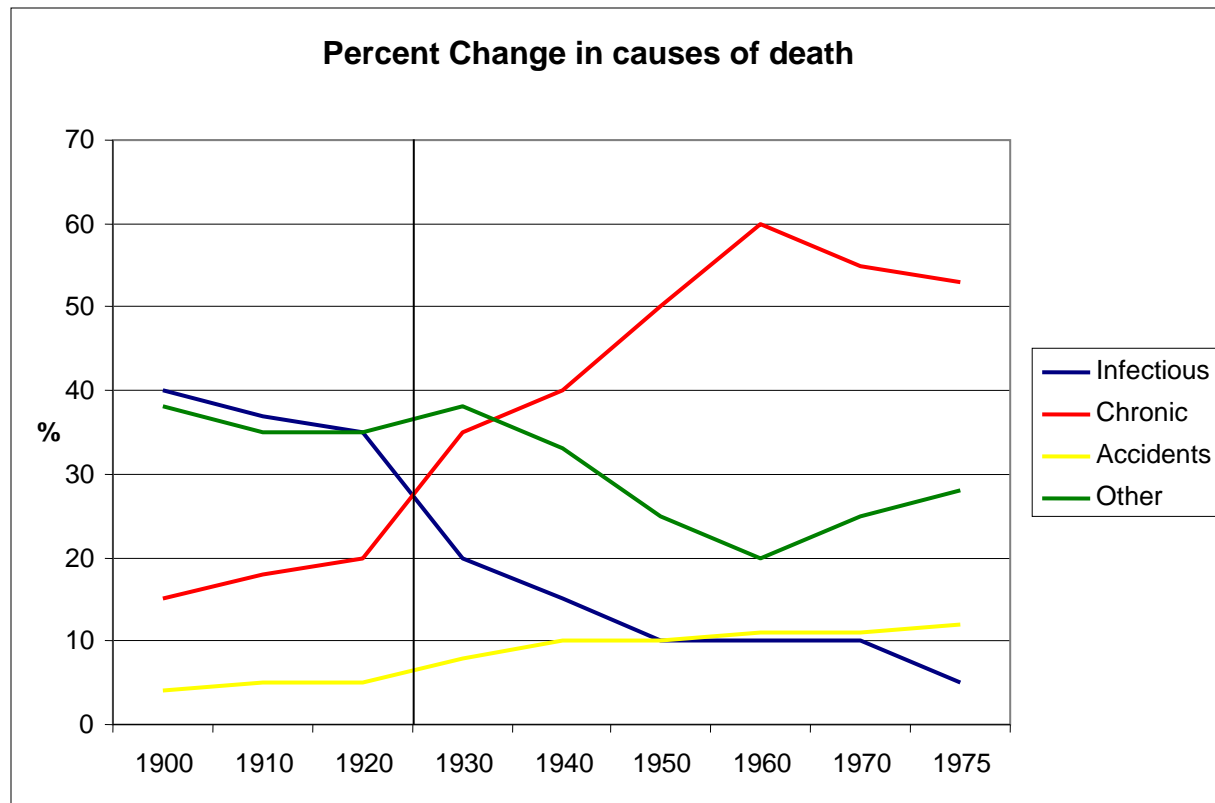
between 1870 and 1900. Between 1900 and 1930, they found tuberculosis mortality decline to be responsible for 19.8% of the total mortality decline.

Salomon and Murray's (2002) examination of the health transition utilized cause-specific age-standardized death rates by sex to determine the proportion that various causes of death contributed to the overall mortality decline. They found the primary causes of the declines in mortality to be reduction in mortality from influenza, pneumonia, and bronchitis, which contributed 24-28% of the decline; respiratory tuberculosis, 11-12%; and diarrheal diseases, 10-11%; other infectious and parasitic diseases, 14-15%. Thus the reduction in deaths due to these diseases accounted for 59-66% of the total mortality decline (Salomon and Murray 2002).

### **The Age of Degenerative and Man-Made Diseases**

This age is marked by the growing preeminence of chronic diseases as the primary causes of death and the continued decline of infectious disease mortality. At first, mortality continued to fall rapidly. Over time, the rate of mortality decline slowed, eventually tapering off in the 1950s (Figure 3-1). This age began around 1920 in the United States, although it is difficult to determine its timing precisely because mortality data were not available for all regions of the United States until 1933 (Omran 1977a; 1977b). During the *Age of Degenerative and Man-Made Diseases*, tuberculosis mortality declined but was still present among the poor and elderly, particularly males over 50 (Omran 1971; 1977b).

Tuberculosis virtually disappeared by 1970, persisting only as a disease of older men, whereas the primary victims in the previous century had been young women. Between 1890 and 1970 tuberculosis and other infectious and infant disease rates decreased significantly in the U.S. (Omran 1977a, 1977b). In addition, starvation became almost nonexistent, and many nutrient deficiency diseases vanished (Omran 1971).



**FIGURE 3-1. Model of the Age of Degenerative and Man-Made Diseases of the Epidemiologic Transition in the U.S. Based on data from McKinlay and McKinlay (1977).**

In a study of the epidemiologic transition in North Carolina between 1915 and 1970, Omran (1975b) found that 60% of the decrease in infectious diseases was due to a decline in tuberculosis. Declines in mortality due to diarrhea and dysentery accounted for an additional 21% of the total decline. The remaining contributing diseases were typhoid, 9.5%; acute respiratory diseases such as pneumonia, bronchitis, and flu, 7.1%; diseases of childhood (croup, measles, rheumatic fever, whooping cough) 4.2%; syphilis, 4%; and malaria and smallpox, 3.7%.

Three categories of morbidity increased dramatically during this stage. Chronic diseases or “diseases of affluence” include allergies, arthritis, malignant neoplasms, emphysema, coronary artery disease, type II diabetes, kidney disease, obesity, and chronic obstructive pulmonary conditions (Barret et al. 1998; Kaplan and Keil 1993; Omran 1977b). A second

category consists of accidents, particularly automobile accidents. The third category includes conditions caused by stress; among these are drug dependency, hypertension, mental illness, peptic ulcers, depression, anxiety, and suicides (Barret et al. 1998; Kim et al. 2002; Omran 1977b). Higher rates of birth defects and mental deficiencies have also been documented (Barret et al. 1998).

From 1890 thru 1970, the U.S. mortality rates for heart disease and cancer were increasing (Omran 1977a, 1977b); as early as the 1920s heart disease became the principal cause of mortality. An extremely low rheumatic-to-arteriosclerotic ratio developed during this period, indicating an increase in heart disease caused by dietary and activity level transitions. By 1940 the diseases most often associated with mortality in modern Western societies—heart disease, stroke, and cancer—had become preeminent and continue to be so today (Omran 1971). By the 1950s, diabetes had replaced tuberculosis on the list of the ten leading causes of death (Omran 1975b). In the 1920s, cancer rates were still low, particularly among those aged 45 years or younger. Though cancer rates remain low for those younger than age 30, cancer is now a major cause of mortality. Cancers of the respiratory system, digestive tract and genitourinary system claim the most lives (Omran 1975b).

The shift from infectious diseases that often kill children to degenerative diseases that primarily afflict the elderly caused life expectancy at birth to increase (Omran 1977b). The increase in life expectancy during this age has contributed to the increase in deaths due to chronic diseases that disproportionately affect the elderly (Alter and Riley 1989). In addition to an aging population during this stage of the transition, fertility decreased and continues to decline slowly. An exception was the baby boom (1946-1964) in the U.S. (Omran 1977a, 1977b).

## Causes of Disease Transition

“Epidemiologic transition” generally refers to the shift from the infectious diseases prevalent in the past to the chronic diseases prevalent among today’s industrialized nations (Armelagos 2004). Most studies of the epidemiologic transition focus on this shift; that is, the transition that leads from the *Age of Receding Pandemics* to the *Age of Degenerative and Man-Made Diseases*. Omran (1977a) identified a number of causes of mortality decline in the 19<sup>th</sup> and 20<sup>th</sup> centuries. He described the classical model of mortality decline as being the result of social, economic, and environmental improvements associated with modernization. The earliest shifts owed little to medical scientific technology, in contrast to those of the delayed model in the 20<sup>th</sup> century, which were caused primarily by medical progress. Better sanitation, quarantines, immunization, and other environmental measures contributed to the decrease in the incidence of infectious diseases in the classical model. Improved treatment and nutrition contributed to a reduction in disease fatality.

The collective impact of these developments may have affected disease patterns as early as the 19<sup>th</sup> century. An improvement in living standards was one such development. The general improvement in nutritional intake reduced mortality by increasing resistance to infection and tolerance to severe illnesses. Personal hygiene improved with socioeconomic development and was a strong barrier against infection. Better housing was also available to more people (Omran 1977b). Other researchers have pointed to factors such as pasteurization, better public sanitation, and improved access to primary care as most responsible for the decline (Kunitz 1991; Woods 1991). Schofield, Reher, and Bideau (1991), in an edited volume on the mortality decline in Europe, concluded that there was no simple cause or explanation for the mortality decline. Mortality decline, they stated, was the result of many different factors.

## **Living Standards and Nutritional Intake**

Popkin (1993, 1994) and McKeown (1983) emphasize diet as a primary cause of the epidemiologic transition. In an examination of the mortality decline in Great Britain between 1848 and 1972, McKeown (1976) suggested that advances in living standards were primarily responsible for the observed improvements in health. McKeown and Record (1962) found likewise that during the 19<sup>th</sup> century the major reason for declining death rates in England and Wales was the increased standard of living. These studies proposed further that associated improvements in nutritional intake were paramount in the European mortality decline. The most dramatic impact of the improvement in diet was a decline in the tuberculosis death rate (McKeown and Record 1962). Popkin (1993) also has linked epidemiologic transition with changes in dietary intake and nutritional quality. He suggested that predictable changes in food intake were a part of the process of modernization and caused shifts in mortality patterns. Popkin has developed a theory of nutrition transition based on five patterns roughly reflecting historical developments: collecting food, famine, receding famine, degenerative diseases, and behavioral change. He related specific socioeconomic factors including diet, economy, demography, and food processing to each pattern. Using these variables, Popkin developed models similar to Omran's three models of epidemiologic transition: the Western High-Income Model; the Japanese and Korean Accelerated Model; and two models for low-income countries: one for those with a rapidly increasing income, and one for those with limited or no economic improvement.

Others researchers contend that the evidence supporting the primacy of diet and nutrition is inconclusive. Wilkinson (1994) suggested that economic growth was the driving force behind all other causes of epidemiologic transition. In their analysis of the transition in Philadelphia

between 1870 and 1930, Condran and Cheney (1982) found economic status to be an important factor in the reduction of diarrheal diseases. Though water quality was also a factor, they found it to be less important than they had expected. In 1930, home-ownership was highly correlated with the number of people living in a home. Home-ownership also was the best predictor of the risk of death due to tuberculosis and was a predictor of contracting pneumonia. Home-owners tended to have better housing conditions and fewer people living in the home. The number of people living in a household had been an important contributor to high tuberculosis and pneumonia prevalence in 1880 (Condran and Cheney 1982).

Whereas both education and personal wealth impact epidemiologic transition by contributing to the reduction in infectious diseases, these may actually have both positive and negative impacts on health. Unhealthy habits and a high-fat, high-calorie diet have been associated with higher discretionary income. Cigarette smoking, greater sedentism, and the consumption of high-fat foods all increased as incomes increased. Thus, the increase in living standards may have contributed to the decrease in infectious diseases while also increasing the incidence of chronic diseases (Pearson 2003).

Between 1900 and 1930 advances in food quality and food science were related to economic growth and technological change. Food preservation through cooling has been implicated in improvements in the type and quality of foods consumed. Cooling and freezing foods retard bacterial growth. By the late 1800s, ice boxes were as common as stoves in most homes. Even in the poorest homes, ice was kept in small quantities (Condran and Cheney 1982). Cooling was not the only method for ensuring a safer food supply. A source of bovine tuberculosis, streptococcal infection and enteric diseases, milk typically had not been sterilized

other than through occasional boiling until pasteurization was introduced as a protective measure in 1909 (Omran 1977b).

That foods could help prevent disease has been recognized for generations. The ancient Egyptians and Greeks utilized animal livers to prevent and treat blindness (Vitamin A deficiency). British scientists discovered that limes could be used to prevent scurvy, a Vitamin C deficiency disease that plagued sailors during the 15<sup>th</sup> and 16<sup>th</sup> centuries. Native Americans prevented scurvy using extracts from pine needles. However the role of vitamins had not been recognized prior to experiments performed in the early 20<sup>th</sup> century. Experiments in 1906 were designed to examine the growth-enhancing substance believed to exist in butterfat and cod liver oil. Casimir Funk coined the term “vitamine” from “vital amine” in the early 1900s. The first vitamin to be discovered was vitamin A in 1913, and another was discovered by 1921. The last of the important vitamins was discovered in 1948. With the discovery of vitamins and the roles they play in nutrition, as well as the identification of foods high in vitamins, health officials could encourage the consumption of particular foods to prevent disease at the same time that people were more able afford them (Elvehjem 1949; Wardlaw and Insell 1996).

### **Public Health and Medical Services**

Mercer (1990) suggested that public health and preventive services were of paramount importance to the decline of infectious diseases and challenged the idea that living standards and nutritional intake were chiefly responsible for the epidemiologic transition. Other researchers suggest that increases in the general wealth of the population may have increased the funds available for public expenditures on health-related efforts, improving overall health of the population (Condran and Cheney 1982). Numerous organized public health services were established in the U.S. during the 19<sup>th</sup> century and the first part of the 20<sup>th</sup> century. The

American Public Health Association was created in 1872. Congress adopted the Eaton Bill which created the National Board of Health in 1879. The precursor to the American Lung Association, the National Association for the Study and Prevention of Tuberculosis, was founded in 1904. Organizations for the prevention of chronic diseases were established later, in the 20<sup>th</sup> century. The American Society for the Control of Cancer was not organized until 1913 and the American Heart Association was not established until 1922 (Omran 1977b).

Public officials during the latter part of the 19<sup>th</sup> century came under increasing pressure to do something about the epidemics ravaging the large cities. Attempts to disinfect the squalid city slums by splashing and spraying carbolic acid throughout the crowded streets did little to curb the outbreaks. These efforts were more of a public relations ploy to convince the citizens that the government was aggressively fighting the problem. Quarantine was another such ploy, since it did little to prevent the spread of diseases such as cholera or yellow fever (Bettmann 1974), although it did provide some relief and treatment for certain other diseases. Isolation of ill patients in fever hospitals and wards served to remove the source of infection and thus protect the uninfected from exposure. Sanatoriums were successful in providing opportunities for patients to get better food and rest. Sanatorium treatment was introduced to deal with tuberculosis, which was most prevalent in poor, overcrowded, dark, poorly-ventilated dwellings. The sanatorium was designed to provide the patient with extended rest, clean air, sunshine, and better nutrition (Omran 1977b; Wilson 1990). By the early 20<sup>th</sup> century, children living in homes with a person sick from tuberculosis were usually removed (Ewbank and Preston 1990).

The effectiveness of public health activities in reducing typhoid fever mortality is well documented: in the latter part of 19<sup>th</sup> century, public health officials identified contaminated water supplies as the source of the disease (Condran and Cheney 1982). In the 20<sup>th</sup> century,



cattle control was added as a means to combat the spread of tuberculosis (Omran 1977b). A variety of public health measures reduced the death rates from diarrheal diseases. In Philadelphia, pamphlets on infant feeding practices were produced for the public. Officials also began to inspect milk for contamination. A child hygiene bureau was established, which opened clinics in those parts of the city that had particularly high mortality rates for infants and children (Condran and Cheney 1982). In their analysis of the epidemiologic transition in Manti, Utah, Levison et al. (1981) found that improvements in public health measures and sanitation, along with the acceptance of medical therapies in place of folk medicines, were responsible for the decline in mortality. Reductions in infectious, parasitic, and respiratory diseases in Utah were a result of a cleaner water supply and modern medical practices.

Improvements in medical and preventive practices ultimately may have been a contributing factor to the epidemiologic transition, but advances in technology during the Gilded Age in the U.S. were not mirrored by advances in the science of medicine. Medical intervention and preventive procedures by 1900 were still sorely lacking. Hospitals of the time were run primarily as charitable institutions utilized only in extreme circumstances by the poor, who were unable to afford home care (Bettmann 1974). In the latter part of the 19th century hospital conditions in health units in Europe and the United States were so poor that Florence Nightingale wrote in *Notes on Hospitals* the famous statement that hospitals "should do the sick no harm" (1863:iii). Doctors of the 19<sup>th</sup> century were not particularly well trained. The profession was one of a nomadic prescriber whose remedies were often completely ineffectual. As late as the 1890s the medical field was often devoid of science, commercially driven, and in many cases a complete sham (Bettmann 1974).

Some aspects of medical care had improved, however, and may have had an impact on health conditions (Omran 1977b). Hospital deliveries of babies increased in frequency during the latter part of the 19th century. Most women still had their children at home, sometimes with the services of a midwife, but with no other health professionals present (Omran 1977b). In 1900, midwives delivered 50 percent of babies in the U.S. but only 20 percent by 1930 (Kobrin, 1966). Extreme criticism of medical training and practice at the end of the 19<sup>th</sup> century also eventually led to reforms (Bettmann 1974). Scientific breakthroughs of the time also led to better pharmaceutical treatments. As the 20<sup>th</sup> century progressed the importance of consulting a doctor in the case of illness was increasingly recognized (Starr 1982).

### **Personal and Household Hygiene**

Few studies have documented the potential impact of personal health care and hygienic practices on reducing mortality. Ewbank and Preston (1990) examined the medical and public health literature for indications that parental health practices were an important factor in changing infant and childhood mortality levels. They also searched for evidence that efforts were made to alter individual health behaviors and to determine if personal hygiene programs actually changed behavior. Some studies of cross-sectional variation in childhood mortality suggested to them that personal health practices might have been more important than previously thought; however, because personal health practices are not as well documented as public health programs or income levels, the importance of these factors may have been overlooked.

By the beginning of the 20th century, mothers had been given the responsibility of protecting their children against disease. The new emphasis on this job for mothers was most likely due to an increasing acknowledgement of the germ theory of disease in the decades after Koch's identification of the tuberculosis bacillus in 1882. Between 1895 and 1930, mothers

received health instruction of two basic types. The first emphasized appropriate nutrition for infants and children. The second stressed the importance of cleanliness and maintaining a hygienic home environment. Hand washing became an important practice for preventing the spread of disease (Ewbank and Preston 1990).

Changes in household hygiene were more likely to be put into practice by members of the upper classes and by those living in the cities, where education was more widely accessible. The differential in child mortality by social class and urban/rural setting increased between 1895 and 1930 in a manner consistent with a more thorough adoption of healthy behaviors by upper-class, urban populations. Ewbank and Preston (1990) believed that this suggested that personal hygiene practices were more likely to be adopted among the urban upper-classes and were responsible for observed declines in infant and child mortality. The authors cited Veiller (1921) to show that although crowding in homes may have become more problematic by 1920, homes were generally more sanitary than they had been in 1870—improvements that were more obvious in urban than in rural homes. Contemporary observers of the time (Abbott 1900; Fox 1919) reported on the poor hygiene often found on rural farms (Ewbank and Preston 1990).

The decline in mortality in developing nations has occurred more recently and is considered unequivocally to be the result of medical interventions. The effects of modern biomedicine have been most significant for and are typical of the contemporary or delayed transition model (Omran 1971). Preston (1975, 1980) also argued that rapid gains in survivorship among people in developing countries following World War II were primarily the result of medical care and public health policies.

## **Immunization**

Though great discoveries in infectious disease transmission were being made by scientists such as Pasteur and Lister during the latter half of the 19<sup>th</sup> century, it would take decades for their scientific principles to be translated into public health practice (Bettmann 1974). Throughout most of the 19<sup>th</sup> century the only immunization available was for protection against smallpox. Inoculation with smallpox virus (variolation) was the primary method of immunization prior to the 1796 development of vaccination by Edward Jenner, using cowpox virus. Variolation was a risky practice, since it could potentially produce a fatal case of smallpox by using the live virus; whereas vaccination was safer and eventually became widespread. The medical and social science literature on smallpox suggests that vaccination was the primary cause of the disease's decline. However, it has not been determined with certainty whether smallpox epidemics in fact ceased at the end of the 19<sup>th</sup> century due to public health efforts to eradicate the disease (Condran and Cheney 1982). Historical studies on the effectiveness of smallpox vaccination in Finland indicated that vaccination was effective at protecting children and increased intervals between outbreaks (Jorde et al. 1990; Mielke et al. 1984).

In 1895, an effective antitoxin for diphtheria was developed, and the decline in diphtheria fatalities at the close of the 19<sup>th</sup> century is attributed by many to the use of this antitoxin (Condran and Cheney 1982). Antitoxin for diphtheria had a significant role in establishing physicians as curers of disease, given that for the first time they effectively were able to cure an otherwise fatal condition (Ewbank and Preston 1990). Success in the elimination of polio and smallpox led to optimism among the modern medical establishment that they would eradicate all infectious diseases by the close of the 20<sup>th</sup> century (Garrett 1994).

## **Changes in Disease Virulence**

The reproductive capacity and brief generation span of disease pathogens make it possible for them to adapt quickly to their host and increase or decrease their virulence (Armelagos 1990; Ewald 1993). Endemicity of a disease in a population is taken as an indication of host and pathogen adaptations that decrease virulence (McNeill 1976). Some researchers have suggested that the reduction of the incidence of plague and other pandemics in Europe was not due to advances in medical science; that prior to 1935, therapeutic interventions had no significant impact, and immunizations had only a small effect on survivorship (McKeown and Brown 1955; McKeown 1976). Ecological reduction in virulence has been implicated in the decreased mortality of some diseases, however. Scarlet fever is one such example. The disease in the past was often fatal, but during the last part of the 19th century through the 20th century the streptococcus pathogen causing the disease became less virulent (Omran 1977b). The decline of scarlet fever thus has been attributed primarily to a reduction in the virulence of the disease agent (McKeown 1976; McKeown and Record 1962; Preston and Van de Walle 1978; Sydenstricker 1933). An evolutionary adaptation of the infective organism to the host was the likeliest source of the decline (Condran and Cheney 1982). Diphtheria may be another such example, since its reduction began before the use of antitoxin (Omran 1977b), although other researchers credit the antitoxin for its decline. Tuberculosis may have declined as a result of populations becoming more resistant to the disease through repeated exposure and genetic selection. Death rates due to pneumonia decreased between 1920 and 1930, preceding the use of antibiotics. Penicillin was not available until 1930, and was not widely used until after the Second World War (Condran and Cheney 1982).

In addition to scarlet fever and diphtheria, decreases in deaths from smallpox, measles, and plague have been attributed to pathogen adaptation. Plague disappeared from Europe in the 1700s. Smallpox and measles became endemic childhood infections as epidemics subsided (Haines 1995; Lancaster 1990). Humans may have adapted to the presence of these pathogens but have been exposed to endemic infectious diseases for only a relatively short generational time. Because of their longer generation time, the genetic adaptation of humans to particular disease organisms is not be as likely as the reverse (Armelagos 1990).

### **Increase in Chronic Disease**

Many analyses of epidemiologic transition view the shift from Omran's second age to the third age likewise to be a result of advances in various aspects of public health, medical technology, nutrition and sanitation. With the reduction in infectious diseases that target the young, more people live long enough to suffer from the degenerative diseases associated with aging (Omran 1975a, 1977b): chronic diseases tend to kill people at older ages. Most researchers interpret the shift as the result of the decline in infectious diseases, of which the rise in chronic diseases is an inevitable consequence. Until recently, few studies had examined the transition as an absolute increase in the prevalence of chronic diseases. One of the few to do so used a simulation model based on dietary intake that predicted significantly higher rates of early death from chronic diseases associated with the Western-style diet than from a broad plant-based diet. The model predicted 2.12 million deaths per year due to a Western diet versus 0.28 million deaths with a traditional Asian diet (Flood 1999).

The dramatic rise of chronic diseases among Native Americans in particular has prompted a number of studies of how and why these diseases have become so prevalent in these populations, beyond simple declines in infectious diseases and longer life expectancy. Because

of the timing of the epidemiological transition among Native American populations, many of the studies focused on the factors that contributed to the recent but rapid increase in mortality due to chronic and degenerative diseases (Manson and Altschul 2004; Lee et al. 1998; Young 1994, 1996).

In their examination of the demographic transition in Philadelphia, Condran and Cheney (1982) found that no individual cause could adequately account for the shift in mortality. They suggested that an adequate model of disease transition would have to account for advances in medicine, alterations in the virulence of infective organisms, shifts in the socioeconomic status of the citizenry, and improvements in sanitation and public health policies. They saw these factors not as rival explanations for the decline in mortality, but as a complex of interrelated sources of the demographic transition.

## **Variation in the Epidemiologic Transition**

### **Socioeconomic Differences**

As more data have been collected regarding the mortality shift, it is less often seen as an all-encompassing occurrence than as a series of discrete events in a population that differ with regard to their timing and causation (Coelho 1997; Condran and Cheney 1982). In industrialized societies, socioeconomic, racial, and sex differences are correlated with variation in morbidity and mortality rates due to infectious and chronic diseases (Arriaga 1989; Blair 1993; Gaylin and Kates 1997). Indeed, social factors operating within societies may have as great an impact on survival as the pathogens themselves. Poverty has been cited as the world's most prolific killer and cause of disease and human suffering (Armelagos 2004; Farmer 2004). Relative income has also been shown to be a strong predictor of health status, and may interact with absolute income as a co-determinant of variation in health status within regions (Rickert 1998). For example (as

discussed earlier in this section), new medical and behavioral procedures for fighting diseases following the acceptance of the germ theory were first put into use by the upper class of the 19th and early 20th centuries (Preston 1985; Sundin 1985).

Housing and living conditions have been implicated in variation in disease epidemiology. The larger houses and fewer children of wealthy families made isolating the sick easier. Women in wealthier families were better positioned to give the necessary time and effort to combat filth and germs in the manner proscribed by health officials (Ewbank and Preston 1990). An analysis of differences in mortality between individuals living in poorhouses and the general population in 19th century New York suggested that these facilities were not an effective strategy for meeting the needs of those living in poverty and contributed to higher mortality rates (Higgins 1998). Today, living in a low socioeconomic status neighborhood has been shown to promote higher mortality independently of low-socioeconomic-status itself (Winkleby and Cubbin 2003). People in the wealthier classes also were more likely to consult physicians when someone in the family became sick (Ewbank and Preston 1990). Large differentials in seeking medical help were recorded between 1921 and 1924. Among the wealthy, 82% saw physicians when suffering from illnesses; among the poor, only 47% sought professional medical treatment (Sydenstricker 1933). By 1930, 60% of children in the wealthiest families had been given a medical assessment, compared with just 40% of children from the poorest families. Diphtheria immunizations had been given to 20% of the wealthiest children and to only 10% of the poorest, as indicated by the White House Conference on Child Protection in 1931 (Ewbank and Preston 1990).

Other researchers point out that poverty and its correlates negatively affect a person's impression of their own empowerment and their appreciation of good health as well as their



ability to seek medical services. They suggest that the poor and the poorly educated have been slower to abandon detrimental health behaviors. Inadequate nutritional intake, smoking, and alcohol abuse are more common among those with lower incomes and less education. Today, as in the past, social class and economic standing correlate with lifestyle and the probability of premature death (Carrigan 1998). Indeed, income has been shown to be strongly and inversely correlated with crude death rates, age-adjusted death rates, premature deaths, infant and adult deaths, natural and non-natural deaths and general levels of health in a population (Rickert 1998). Contemporary researchers suggested that poor and rural populations were less likely to alter their habits and thus continued to suffer greater morbidity and mortality.

The indication is that variation in personal health care practices from 1900-1930 were a significant factor in differences in infant and child mortality. Ewbank and Preston (1990) researched the efficacy of new home hygiene and childcare practices and examined differentials in child mortality between people in different social classes during the early 20th century. They suggested that the observed decline in child mortality between 1900 and 1925 was the result of more modern health care and childcare practices. They saw decreasing child mortality among professionals in the early 20th century, supporting the hypothesis that well-educated professionals were the first to accept new child care and hygiene practices designed to reduce illness. The children of railroad laborers however did not experience the rapid decrease in child mortality that the children of other skilled workers in the industry experienced. In the farming industry, farm workers experienced child mortality levels 20-30% higher than farmers, whereas farm foremen experienced child mortality rates that were 10-15% below that of the farmers. In this analysis however, a more rapid decrease in mortality among professional and managerial

groups was explained by urbanization level and ethnicity; income was not a major aspect of the differential in child mortality in this group (Ewbank and Preston 1990).

In some cases, an urban or rural location seems to affect the disease transition in a manner similar to wealth. In England in 1922 urban areas had lower infant mortality levels than did rural areas. This was perhaps due to education on infant care being more widely available in the cities. Because rural areas were more isolated, less information was available to these people, resulting in greater ignorance with regard to innovations in infant care (Ewbank and Preston 1990). Medical services also were more accessible to urban dwellers. By 1930, vaccinations were given to 21% of urban U.S. children, but only to 7% of rural children (Wilbur et al. 1931; Ewbank and Preston 1990). In their analysis of the 1900 PUS (Public Use Sample of the U.S. Census), Preston and Haines (1989) found urbanization and ethnicity to be among the most significant factors in childhood deaths. Nonetheless, cities did present unique challenges to disease eradication. The perseverance of high rates of tuberculosis in cities has been recorded. Preston and Van de Walle (1978) documented persistent and increasing death rates from tuberculosis in 19th century Paris, although declines in mortality were recorded for typhoid, cholera, and other diarrheal diseases that primarily spread through contaminated water.

Regional variation in disease prevalence was common in America. Pellagra, a disease caused by a niacin deficiency (a member of the B vitamin complex) and typified by dermatitis, diarrhea, delirium, and ultimately, death, was more common in the rural South than in the urbanized North during the early part of the 20th century (Omran 1977b). A geographic and cultural analysis of mortality patterns in North Carolina found significant geographic differences in susceptibility to infant mortality, general mortality, influenza and pneumonia, tuberculosis, cancer and heart diseases. The variation was consistent with the epidemiologic transition theory,

which predicts that more economically advanced regions go through the transition earlier and more quickly (Rice 1983).

Western nations were not the only ones to see variation within their borders with regard to the kinds of diseases contributing to mortality. Variation within a single nation has been identified as a common characteristic of countries undergoing the epidemiologic transition in Latin America. Whereas chronic diseases have become a public health problem among many nations of Central and South America, infectious diseases persist as a significant cause of mortality (Frenk et al. 1991; Guerra-Godinez et al. 2003; Prata 1992; Trevino Garcia-Manzo et al. 1994).

### **Variation by Race, Ethnicity, and Gender**

A mortality decline was experienced by European Americans earlier than by minorities; thus the epidemiologic transition among minority groups in the U.S. occurred after it had already taken place among European Americans. Americans of European descent generally enjoyed better socioeconomic conditions and better housing, educational opportunities, nutrition, and access to health care. For whites, the epidemiologic transition in mortality and fertility began at an earlier time and progressed more rapidly. Life expectancy among whites also increased more quickly than among non-whites (Omran 1977a, 1977b). Studies of racial and socioeconomic differences on mortality indicated that as these differences fluctuated, so did mortality rates (Potter 1989).

Minority death rates from tuberculosis have always been greater than those of whites, particularly among young adults and children. During the first half of the 20th century, minorities between the ages 20 and 60 years suffered and died from tuberculosis at disproportionately high rates, although by the 1970s the risk of death due to tuberculosis had

virtually disappeared (Omran 1975b). More minorities still die of bronchitis, diarrhea, enteritis, pneumonia, other infectious diseases and diseases of early childhood more often than do whites (LaVeist 2006; Omran 1977b). The rate of maternal mortality among minority groups in the past was much higher than that of the white women (Omran 1975a, 1977a, 1977b).

Just as there has been variation in the timing and pattern of the epidemiologic transition among racial groups, so is there interracial variation in the incidence of particular chronic diseases today. Heart disease is more common among whites than among Asian or African Americans and is now most common among Native Americans. Also higher among Native Americans are strokes, which are comparatively high among blacks as well, among whom the highest rates of high blood pressure are reported. Hispanics, Native Americans, and African Americans all have higher diabetes rates than do European or Asian Americans (Barnes et al. 2005; NCHS 2005). Among the various racial groups, African Americans and Native Americans are more likely to suffer due to injuries. Hispanics are less likely than non-Hispanics to die from injuries, though some types of injuries, such as pedestrian fatalities, are more common among Hispanic people (NCIPC 2001). Though significant improvements in maternal mortality rates have been made over the years, certain racial and ethnic groups have not made as substantial gains as others; in particular, African Americans when compared with European Americans (Anachebe 2006).

In the 1920s, cancer was uncommon among those under the age of 45 and lower among whites than minorities. However in more recent decades whites have had a higher burden for many types of cancer (Omran 1975b). Despite a lower incidence of breast cancer among African American women compared to European American women, the former are 33% more likely to die from the disease than the latter (NCHS 2004).

Within racial groups, females have lower mortality rates than males. Among both whites and minorities, infant mortality is greater for males than for females (Omran 1975a, 1977a; 1977b). Elman and Myers (1997), using the 1880 Public Use file from the U.S. census, discovered significant differences in morbidity between sexes in early adulthood and old age. Males historically have a much higher mortality rate than females from unintentional injuries (Relethford 1991).

Compared to other industrialized nations, the U.S. consistently has had a higher infant mortality rate, which may be due to the ethnic and racial variability of the U.S. population. A significant proportion of the U.S. citizenry belongs to minority groups who have been slow to achieve the higher socioeconomic status associated with low infant mortality (Omran 1977a, 1977b). Differences in infant mortality rates between racial and ethnic groups in the U.S. thus persist (Anachebe 2006), which also contributes to variability in life expectancy. That said, differences in life expectancy have decreased between Native Americans and whites in the U.S. In 1940, the difference was 10.8 years among males and 14.7 years among females. By 1980, the gap was 3.6 years for males and 3.0 years for females. The major contributor to the differences between the two groups was infant mortality. Narrowing of the life expectancy gap over the 40-year period has been due primarily to a reduction in childhood deaths among Native Americans. Native Americans who live into adulthood now have a life expectancy similar to most other U.S. populations (Young 1994). A recent analysis of variation in life expectancy in the U.S. identified race, location, income, population density, and local homicide rates as the most important factors regarding the expectation of life at birth (Murray et al. 2006).

## **Genetic Influences on Variation in Mortality and Disease susceptibility within Populations**

Racial and ethnic differences in disease resistance in particular suggest the possibility that genetics plays a role in disease susceptibility. Major epidemics in history were certainly widespread enough to affect gene frequencies: the 14th century bubonic plague epidemic in Europe and the 1918-1919 Spanish flu pandemic killed large segments of populations; cholera has been associated with blood type O in India; and malaria is known to have selected for the allele that causes sickle cell anemia (Svanborg-Eden and Levin 1990). Differential history and prehistory of exposure to diseases caused differences in susceptibility when long-separated populations came into contact (Ramenofsky 1993; Kunitz 1993). Evidence suggests that variation in the major histocompatibility complex may impact disease susceptibility (Coelho 1997). Thus it is possible that different ethnic and racial groups within the same environment and population will have variable genetic susceptibilities to pathogens (Svanborg-Eden and Levin 1990).

Despite genetic contributions to differential disease mortality, most of the variation in infectious disease rates is likely to be environmental in nature, particularly among modern populations. Evidence strongly suggests that the ecosystem, sanitary conditions, lifestyle, and other cultural factors have an overwhelming impact on infectious disease rates (Svanborg-Eden and Levin 1990). Most risk factors for chronic diseases such as cardiovascular disease, however, show both biological and cultural influences: high blood pressure, abnormal cholesterol levels, type 2 diabetes, and obesity all correlate with biology and behavior (Sharma et al. 2004). One other significant risk factor – smoking – seems primarily behavioral, though studies suggest that smoking also has a biological component (Brody et al. 2006; Shields et al. 1998). Among modern U.S. populations with below average socioeconomic standing, African Americans

exhibit more risk factors for cardiovascular disease. Among those with a higher socioeconomic status, both African Americans and non-white Hispanics exhibit significantly more cardiovascular disease risk factors (Sharma et al. 2004).

Recent studies indicate that race and socioeconomic status are still major determinants of health status. Modern epidemiological investigations generally measure socioeconomic status by quantifying education level, occupation, and income (Kaplan and Keil 1993). Unlike the situation among European Americans, however, African Americans do not show marked improvements in health status with increased education. This indicates the more prominent roles played by race and income rather than education or employment (Farmer and Ferraro 2005). However, the manner in which race contributes to mortality is still unclear. A 1993 issue of the CDC's Morbidity and Mortality Weekly Report concluded that neither race nor ethnicity were themselves risk factors for death, but markers used to better comprehend real risk factors (CDC 1993).

## **Epidemiologic Transition among Native American Groups**

With the exception of the last 50 years, data on health and disease among Native Americans is poor, even in the historic period. The official statistics and records were incomplete, inconsistent, and often unreliable (Young 1994). Thus, few studies on the epidemiologic transition among Native American groups have been undertaken.

One thorough analysis was Broudy and May's (1983) examination of the epidemiologic transition among the Navajo (Diné). The previous decades had seen dramatic changes in the lives of the Navajo. The demographic and epidemiologic changes reflected the rapid social and economic changes, on the Navajo reservation after 1960 (Broudy and May 1983). The Dine had shifted from a more traditional subsistence economy to an increasing tendency to rely more on

working for a paycheck. The household and family structure among the Navajo had been changing from an extended family arrangement to the nuclear family more common among the rest of the U.S. (Kunitz and Levy 1981). An adequate supply of food from stores and government commodities had become more accessible. The consumption of high-calorie, low-quality foods rapidly became common and developed into a primary concern of many public health experts (Broudy and May 1983). In addition to modern foods, the use of Western medical services was becoming standard practice among the Navajo by 1980 (Stewart et al. 1980).

The process of modernization impacted fertility levels among the Navajo. Their fertility rate (General Fertility Rate) decreased from 55.4 to 33.1 between 1965 and 1978. The infant mortality rate also dropped during this time period, from 52 per 1,000 births to just 15.2. Despite the overall lower infant mortality rate, the death rate among post-neonates was still significantly impacted by infectious diseases (Broudy and May 1983).

In the early 1980s, the Navajo were much more economically disadvantaged than the rest of the U.S. population, but by this time, crude death rates among the Navajos had decreased to a lower level than that of the U.S. population in general. However, the lower death rate among the Navajo was due to their much younger population. The age-adjusted death rates indicated that Navajo mortality was almost 50% higher than that among the general U.S. population (Broudy and May 1983).

The most serious health problems among the Navajo were associated with social behaviors and the environment. The primary cause of mortality was accidents in motor vehicles. By the 1970s, motor vehicle accidents were subtracting 5.2 years of life from male Navajo and 2.7 years of life from female Navajo (Carr and Lee 1978). Alcohol abuse had become a serious contributor to mortality as well, with related liver cirrhosis, homicides, and suicides increasing in



frequency and becoming major causes of mortality. Navajo men were experiencing very different mortality characteristics than their female counterparts or other U.S. males of comparable age. Half of all male deaths were due to social or behavioral factors. Only 27% of deaths among females were due to the same factors (Broudy and May 1983). This pattern of mortality continued for the Navajo into the 1990s (Howard 1991).

Accidents had become the primary cause of death followed by heart disease, cancer, influenza, and pneumonia. Nutritional and most infectious diseases other than influenza and pneumonia were no longer significant among the Navajo. The authors concluded that by the 1980s the Navajo had passed through the *Age of Pestilence and Famine* and were in the latter stages of the *Age of Receding Pandemics*, somewhat straddling Stages Two and Three as described by Omran. The dramatic rise in behavior-related deaths was seen as additional evidence of an epidemiologic transition among the Navajo, albeit one with its own particular characteristics. The health care system was imposed upon the Navajo from the outside, thus its effects were independent of the other factors associated with modernization. Infectious diseases were easily cured therapeutically and thus they were quickly eliminated as major causes of mortality. Fertility however was not so easily lowered thus leading to a young population. This pattern seems more common when health care methods and technology are imposed from the outside. Among the Navajo, with rapid modernization it appeared that behaviorally influenced conditions and deaths increased much more quickly than did chronic diseases, leading to a pattern of epidemiologic transition that differed markedly from that of the greater U.S. population (Broudy and May 1983; Stull 1972).

Trafzer (1997) studied the epidemiologic transition among the Yakama in Washington. This analysis tracked the epidemiologic transition from 1888 to 1964, chronicling the effects of

reservation life on mortality. Reservation agents discouraged the traditional food collection methods of the Yakama, which featured seasonal nomadic gathering of fruits, nuts, and berries, hunting antelope and deer, and fishing for sturgeon and salmon. Instead the government forced the Indians to adopt agriculture and ranching as a means of indoctrinating them into the larger American society. This settlement into permanent housing facilitated the spread of pathogens that killed the Yakama at high rates. The reservation homes were cramped wooden structures very different from their traditional mat lodges. The new housing placed people together at close quarters and with little knowledge of sanitary and hygienic practices that would prevent disease. The three most common causes of death among the Yakama were tuberculosis, pneumonia, and heart disease. Compared to non-Indian populations, the crude rates of death from tuberculosis and pneumonia were many times greater among the Yakama. They also experienced disproportionately high rates of death from gastrointestinal disorders, accidents, influenza, and premature births. Fetal, infant, and childhood deaths were also much higher among the Yakama than among other U.S. populations (Trafzer 1997).

Indian blood quantum correlated with death rates. Most significantly, full bloods died more often from the flu, and new infectious viruses seemed to have caused greater mortality among full-blooded Indians than among those of mixed ancestry. Yakama full bloods also died more often from automobile accidents. However with regard to the more degenerative diseases of heart disease and cancer, those of mixed Indian ancestry made up a greater percentage of the dead (Trafzer 1997).

The Yakama infant mortality rate peaked in 1929 at ten times that of the rest of the state of Washington. By 1964 infant mortality among the Yakama had dropped dramatically, but was still double that of the surrounding population. The crude death rate of the entire Yakama

population peaked in 1940 at over 60 deaths per 1,000 population. Between 1950 and 1964, the leading cause of death was heart disease, followed closely by pneumonia. Accidents and then cancer had become the other major causes of mortality (Trafzer 1997).

Another tribe-specific analysis of the epidemiologic transition focused on the Blackfeet of northern Montana. This PhD dissertation specifically examined health among females during the 20<sup>th</sup> century, primarily through a dietary analysis, community survey, and interviews with women representing four separate generations. Behavioral changes as they related to changes in the sociopolitical conditions of the Blackfeet constituted the framework for the analysis. The author found that lifestyle changes, particularly those associated with dietary intake and food choices, played a significant role in the health status and disease profile among the women. Biological and behavioral changes included secular trends in height, weight, obesity, fertility, and breast-feeding patterns that were consistent with those among other populations undergoing the epidemiologic transition (Johnston 1999).

A few studies of disease transition have been conducted among Canada's First Nations peoples. These indicate a pattern similar to that documented among Native Americans. Herring and Hoppa (1997) examined three death cohorts among the Western James Bay Cree spanning 1851-1906, 1914-1945, and 1946-1964. They uncovered a seasonal pattern to mortality among the 1851-1906 cohort with a major peak during the summer months and a minor peak during winter. The seasonal pattern of mortality disappeared after the turn of the century. Among the Western James Bay Cree, it appeared that the process of epidemiologic transition began prior to World War II. Social and economic changes that included a decline in the fur trade were implicated in the early transition. Marrett and Chaudhry's (2003) analysis of cancer among Canadian First Nations people indicated that the trends in cancer were consistent with a

population experiencing the epidemiologic transition to the stage of chronic and degenerative diseases. Trovato (1988) compared the 1951-1971 mortality rates and causes among Canadian Indians with the British and French in Canada. The Indians lagged behind the other two groups, with rates indicative of a population undergoing the epidemiologic transition.

These studies suggest that native peoples have experienced the transition to Omran's *Age of Degenerative and Man-Made Diseases* but that the timing and pattern of that transition is particular to Native Americans. Specific anomalies associated with Native Americans include extremely high rates of diabetes, significantly higher rates of mortality due to social pathologies and high rates of alcoholism, liver disease and some cancers.

## **Conclusion**

Epidemiologic Transition theory describes general patterns of mortality over time. Variations in patterns between societies are due to a multitude of important factors impacting societies. In the U.S., various minority groups display mortality patterns that are specific to their particular historical and socioeconomic circumstances. Native Americans, while showing variation along tribal and regional lines, appear to display a distinctive pattern that features a recent and dramatic rise of chronic conditions, a persistence of infectious diseases at higher than normal levels, an extreme dietary shift, and significant social pathologies that reflect Native American historical circumstances.

## **Chapter 3 References Cited**

- Abbott, S.W. 1900. *The Past and Present Condition of Public Hygiene and State Medicine in the United States*. Boston: Wright and Potter Printing Company.
- Alter, G. and Riley, J.C. 1989. Frailty, Sickness, and Death: Models of Morbidity and Mortality in Historical Populations. *Population Studies* 43(1):25-45.
- Anachebe, N.F. 2006. Racial and Ethnic Disparities in Infant and Maternal Mortality. *Ethnicity and Disease* 16(2 S3):S3-71 – S3-76.

Armstrong, G.J. 1990. "Health and Disease in Prehistoric Populations in Transition," in *Disease in Populations in Transition: Anthropological and Epidemiological Perspectives*. Edited by A. Swedlund and G. Armstrong, pp. 124-142. South Hadley: Bergin and Garvey.

—. 2004. "Emerging Disease in the Third Epidemiological Transition," in *The Changing Face of Disease: Implications for Society*. Edited by N. Mascie-Taylor, J. Peters, and S. McGarvey, pp. 7-22. Society for the Study of Human Biology Series: 43. Boca Raton, FL: Routledge.

Armstrong, G.J., Barnes, K.C., and Lin, J. 1996. Disease in Human Evolution: The Re-Emergence of Infectious Disease in the Third Epidemiologic Transition. *National Museum of Natural History Bulletin for Teachers*. 18(3): 1-7.

Arriaga, E.E. 1989. "Changing Trends in Mortality Decline During the Last Decades," in *Differential Mortality: Methodological Issues and Biosocial Factors*. Edited by L. Ruzicka, G. Wunsch, and P. Kane, 1:105-129. Oxford, UK: Clarendon.

Barnes, P.M., Adams, P.F., and Powell-Griner, E. 2005. *Health Characteristics of the American Indian and Alaska Native Adult Population: United States, 1999 – 2003. Advance Data from Vital and Health Statistics, No. 356*. Hyattsville, MD: National Center for Health Statistics.

Barret, R., Kuzawa, C.W., McDade, T., and Armstrong, G.J. 1998. Emerging and Re-emerging Infectious Diseases: The Third Epidemiologic Transition. *Annual Reviews in Anthropology* 27:247-271.

Bettmann, O.L. 1974. *The Good Old Days – They Were Terrible!* New York: Random House.

Blair, A. 1993. "Social Class and Contextualization of Illness Experience," *Worlds of Illness: Biographical and Cultural Perspectives on Health and Disease*. Edited by A. Radley, pp. 114-147. New York: Routledge.

Brody, A.L., Mandelkern, M.A., Olmstead, R.E., Scheibal, D., Hahn, E., Shiraga, S., Zamora-Paja, E., Farahi, J., Saxena, S., London, E.D., and McCracken, J.T. 2006. Gene Variants of Brain Dopamine Pathways and Smoking-Induced Dopamine Release in the Ventral Caudate/Nucleus Accumbens. *Archives of General Psychiatry* 63(7): 808-816.

Broudy, D.W. and May P.A. 1983. Demographic and Epidemiologic Transition among the Navajo Indians. *Social Biology* 30(1): 1-16.

Carr, B.A., and Lee, E.S. 1978. Navajo Tribal Mortality: A Life Table Analysis of the Leading Causes of Death. *Social Biology* 25:279-287.

Carrigan, J.A. 1998. *Inequality, Health Behaviors, and Mortality: A Sociodemographic Analysis*. PhD Dissertation, University of Colorado at Boulder.

CDC (Centers for Disease Control and Prevention). 1993. Use of Race and Ethnicity in Public Health Surveillance: Summary of the CDC/ATSDR Workshop. *Morbidity and Mortality Weekly Report* 42: 1-17.

Coelho, P.R.P. 1997. Epidemiology and the Demographic Transition in the New World. *Health Transition Review* 7(2):237-240.

- Condran, G.A., and Cheney, R.A. 1982. Mortality Trends in Philadelphia: Age- and Cause Specific Death Rates 1870-1930. *Demography* 19(1): 97-123.
- Elman, C. and Myers, G. 1997. Age- and Sex-Differentials in Morbidity at the Start of an Epidemiologic Transition: Returns from the 1880 U.S. Census. *Social Science and Medicine* 45(6): 943-956.
- Elvehjem, C.A. 1949. Seven Decades of Nutrition Research. *Science* 109(2832): 354-358.
- Ewald, P.W. 1993. The Evolution of Virulence. *Scientific American* 268(4):86-93,
- Ewbank, D.C. and Preston, S.H. 1990. "Personal Health Behavior and the Decline in Infant and Child Mortality, The United States, 1900 – 1930," in *What We Know About Health Transition: The Cultural, Social and Behavioral Determinants of Health*. Edited by J.C. Caldwell, S. Findley, P. Caldwell, G. Santow, W. Cosford, J. Braid, and D. Broers-Freeman, pp. 116-149. Canberra: Health Transition Centre, Australian National University.
- Farmer, M.M. and Ferraro, K.F. 2005. Are Racial Disparities in Health Conditional on Socioeconomic Status? *Social Science and Medicine* 60(1):191-204.
- Farmer, P.E. 2004. An Anthropology of Structural Violence. *Current Anthropology* 45(3):305-325.
- Flood, A.P. 1999. *The Construction and Assessment of a Simulation Model to Describe the Impact of Dietary Change on the Epidemiologic Transition and its Consequences in China*. PhD Dissertation, Cornell University.
- Fox, E.G. 1919. "Rural Problems," *Children and Youth: Social Problems and Social Policy*. Edited by W. Chenery and E. Merrit, pp. 186-194. Reprint of a copy from the University of Illinois Library, 1974. Arno Press, Inc.
- Frenk, J., Frejka, T., Bobadilla, J.L., Stern, C., Lozano, R., Sepulveda, J., and Jose, M. 1991. The Epidemiologic Transition in Latin America. *Boletin de la Oficina Sanitaria Panamericana. (Pan American Sanitary Bureau)* 111(6):485-496.
- Garrett, L. 1994. *The Coming Plague: Newly Emerging Diseases in a World out of Balance*. New York: Farrar Straus and Giroux.
- Gaylin, D.S., and Kates, J. 1997. Refocusing the Lens: The Epidemiologic Transition Theory, Mortality Differentials, and the AIDS Pandemic. *Social Science and Medicine* 44(5):609-621.
- Guerra-Godinez, J.C., Larrosa-Haro, A., Coello-Ramirez, P., Tostado, H.R., Riviera-Chavez, E., Castillo de Leon, Y.A., Bojorquez-Ramos Mdel, C., Aguilar-Benavides, S. 2003. Changing Trends in Prevalence, Morbidity, and Lethality in Persistent Diarrhea of Infancy during the Last Decade in Mexico. *Archives of Medical Research* 34(3):209-213.
- Haines, M.R. 1995. "Disease and Health through the Ages," in *The State of Humanity*. Edited by J. Simon, pp. 51-60. Oxford: Basil Blackwell.

- Herring, D.A. and Hoppa, R.D. 1997. Changing Patterns of Mortality Seasonality among the Western James Bay Cree. *International Journal of Circumpolar Health* 56(4):121-133.
- Higgins, R.L. 1998. *The Biology of Poverty: Epidemiological Transition in Western New York*. PhD Dissertation, State University of New York at Buffalo.
- Howard, C.A. 1991. *Navajo Tribal Demography, 1983-1986, in Comparative and Historical Perspective*. PhD Dissertation. The University of New Mexico.
- Johnston, S.L. 1999. *Health and Lifestyle Change among Blackfeet Women of Northern Montana*. PhD Dissertation. University of Pennsylvania.
- Jorde, L.B., Pitkanen, K., Mielke, J.H., Fellman, J.O., and Eriksson, A.W. 1990. "Historical Epidemiology of Smallpox in Kitee, Finland," in *Disease in Populations in Transition: Anthropological and Epidemiological Perspectives*. Edited by A. Swedlund and G. Armelagos, pp. 183-200. New York: Bergin and Garvey.
- Kaplan, G. and Keil, J. 1993. Socioeconomic Factors and Cardiovascular Disease: A Review of the Literature. *Circulation* 88(4):1973-1998.
- Kim, Y.H., Lee, J.H., Lee, S.S., Cho, E.Y., Koh, K.C., Paik, S.W., Rhee, J.C., Choi, K.W., Son, H.J., Rhee, P.L., Kim J.J., and Oh, Y.L. 2002. Long-term Stress and *Helicobacter pylori* Infection Independently Induce Gastric Mucosal Lesions in C57BL/6 Mice. *Scandinavian Journal of Gastroenterology* 37(11):1259-1264.
- Kobrin, F.E. 1966. The American Midwife Controversy: A Crisis of Professionalization. *Bulletin of the History of Medicine* 40:350-363.
- Kunitz, S.J. 1991. "The Personal Physician and the Decline of Mortality," in *The Decline of Mortality in Europe*. Edited by R. Schofield, D. Reher and D. Bideau, pp. 248-262. Oxford, UK: Clarendon.
- . 1993. "Diseases and Mortality in the Americas Since 1700," in *The Cambridge World History of Diseases*. Edited by K. Kiple, pp. 328-333. New York: Cambridge University Press.
- Kunitz, S.J., and Levy, J.E. 1981. "Navajos," *Ethnicity and Medical Care*. Edited by A. Harwood, pp. 337-395. Cambridge: Harvard University Press.
- Lancaster, H.O. 1990. *Expectations of Life: A Study in the Demography, Statistics, and History of World Mortality*. New York: Springer – Verlag.
- LaVeist, T.A. 2006. *Minority Populations and Health: An Introduction to Health Disparities in the United States*. San Francisco: Jossey-Bass.
- Lee, Elisa T., Cowan, Linda D., Welty, T.K., Sievers, M., Howard, William J., Oopik, A., Wang, W., Yen, J., Devereux, R.B., Rhoades, E.R., Fabsitz, R.R., Go, O., and Howard, B.V. 1998. All-Cause Mortality and Cardiovascular Disease Mortality in Three American Indian Populations, Aged 45-74 Years, 1984-1988: The Strong Heart Study. *American Journal of Epidemiology* 147(11):995-1008.

Levison, C.H., Hastings, D.W., and Harrison, J.N. 1981. Epidemiological Transition in a Frontier Town - Manti, Utah: 1849 - 1977. *American Journal of Physical Anthropology* 56:83-93.

Manson, S.M. and Altschul, D.B. 2004. *Cultural Diversity Series: Meeting the Mental Health Needs of American Indians and Alaska Natives*. National Technical Assistance Center for State Mental Health Planning (NTAC), and the National Association of State Mental Health Program Directors (NASMHPD). Rockville, MD: U.S. Department of Health and Human Services.

Marrett, L.D. and Chaudhry, M. 2003. Cancer Incidence and Mortality in Ontario First Nations, 1968 - 1991. *Cancer Causes and Control* 14(3):259-268.

McKeown, T. 1976. *The Modern Rise of Population*. New York: Academic Press.

McKeown, T. 1979. *The Role of Medicine: Dream, Mirage or Nemesis?* Oxford: Basil Blackwell.

—. 1983. Food, Infection, and Population. *Journal of Interdisciplinary History* 14:227.

McKeown, T. and Brown, R.G. 1955. Medical Evidence Related to English Population Change in the Eighteenth Century. *Population Studies* 9:119-141.

McKeown, T. and Record, R.G. 1962. Reasons for the Decline of Mortality in England and Wales During the Nineteenth Century. *Population Studies* 15(2):94-122.

McKinlay, J.B. and McKinlay, S.M. 1977. The Questionable Contribution of Medical Measures to the Decline of Mortality in the United States in the Twentieth Century. *The Milbank Memorial Fund Quarterly. Health and Society* 55(3):405-428.

McNeill, W.H. 1976. *Plagues and People*. Garden City, NY: Academic Press.

Mercer, A. 1990. *Disease Mortality and Population in Transition: Epidemiological – Demographic Change in England since the Eighteenth Century as Part of a Global Phenomenon*. Leicester: Leicester University Press.

Mielke, J.H., Jorde, L.B., Trapp, P.G., Anderton, D.L., Pitkanen, K., and Eriksson, A.W. 1984. Historical Epidemiology of Smallpox in Aland, Finland: 1751 – 1890. *Demography* 21:271-295.

Murray, C.J.L., Kulkarni, S.C., Michaud, C., Tomijima, N., Bulzacchelli, M.T., Iandiorio, T.J., and Ezzati, M. 2006. Eight Americas: Investigating Mortality Disparities across Races, Counties, and Race-Counties in the United States. *PLoS Medicine*. 3(9):e260. DOI: 10.1371/journal.pmed.0030260.

NCHS (National Center for Health Statistics). 2004. *Health, United States 2004: With Chartbook on Trends in the Health of Americans*. Hyattsville, MD: U.S. Department of Health and Human Services.

—. 2005. *Health, United States 2005: With Chartbook on Trends in the Health of Americans*. Hyattsville, MD: U.S. Department of Health and Human Services.

NCIPC (National Center for Injury Prevention and Control). 2001. *Injury Fact Book 2001 – 2002*. Atlanta, GA: Centers for Disease Control and Prevention.



- Nightingale, F. 1863. *Notes on Hospitals*. London: Longman, Green, Longman, Roberts, and Green.
- Notestein, F. 1945. "Population—The Long View," in *Food for the World*. Edited by P. Schultz, pp. 36-57. Chicago: University of Chicago Press.
- Omran, A.R. 1971 The Epidemiologic Transition; A Theory of the Epidemiology of Population Change. *The Milbank Memorial Fund Quarterly* 49(4):509-538.
- . 1975a. The Epidemiologic Transition in North Carolina during the Last 50 to 90 Years, I. The Mortality Transition. *North Carolina Medical Journal* 36(1):23-28.
- . 1975b. The Epidemiologic Transition in North Carolina During the Last 50 to 90 Years: II. Changing Patterns of Disease and Causes of Death. *North Carolina Medical Journal* 36(2):83-88.
- . 1977a. A Century of Epidemiologic Transition in the United States. *Preventive Medicine* 6(1):30-51.
- . 1977b. Epidemiological Transition in the United States: The Health Factor in Population Change. *Population Bulletin* 32:3-42.
- . 1983. The Epidemiologic Transition Theory. A Preliminary Update. *Journal of Tropical Pediatrics* 29(6):305-316.
- Pearson, T.A. 2003. Education and Income: Double-Edged Swords in the Epidemiologic Transition of Cardiovascular Disease. *Ethnicity & Disease* 13(2 suppl):S158-S163.
- Pitkanen, K. 2002. Early Mortality Decline in Norway in Comparative Perspective. *Historical Studies in Mortality Decline*. Det Norske Videnskaps – Akademi. Oslo: Novus Forlag. 11-22.
- Popkin, B.M. 1993. Nutrition Patterns and Transitions. *Population and Development Review* 19:138-157.
- . 1994. The Nutrition Transition in Low-Income Countries: An Emerging Crisis. *Nutrition Reviews* 52(9):285-298.
- Potter, L.B. 1989. *Proximate and Nonproximate causes of Racial Life Expectancy Differentials in the U.S., 1970 - 1980*. PhD Dissertation, The University of Texas at Austin.
- Prata, Pedro R. 1992. The Epidemiological Transition in Brazil. [A transicao epidemiologica no Brasil.] *Cadernos de Saude Publica* 8(2):168-75.
- Preston, S.H. 1975. The Changing Relation between Mortality and Level of Economic Development. *Population Studies* 29(2):231-248.
- . 1980. Causes and Consequences of Mortality Declines in Less Developed Countries during the Twentieth Century. R. Esterlin, ed. *Population and Economic Change in Developing Countries*. Chicago: University of Chicago Press. 289-341.
- . 1985. Resources, Knowledge, and Child Mortality: A Comparison of the U.S. in the Late 19th Century and Developing Countries Today. *Proceedings of the International Union for the Scientific*

- Study of Population General Conference. International Population Conference, Florence, Italy, 5-12 June 1985. *Oridna, Liege* 4:373-386.
- Preston, S.H. and Haines, M. 1989. *Fatal Years: Child Mortality in Late Nineteenth Century America*. Chicago: University of Chicago Press.
- Preston, S.H. and Van de Walle, E. 1978. Urban Trend Mortality in the Nineteenth Century. *Population Studies* 32(2):275-297.
- Puranen, B. 1991. "Tuberculosis and the Decline of Mortality in Sweden," in *The Decline of Mortality in Europe*. Edited by R. Schofield, D. Reher and D. Bideau, pp. 68-96. Oxford, UK: Clarendon.
- Ramenofsky, A. 1993. "Diseases of the Americas, 1492 – 1700," in *The Cambridge World History of Diseases*. Edited by K. Kiple, pp. 417-727. New York: Cambridge University Press.
- Relethford, J.H. 1991. Sex Differentials in Unintentional Injury Mortality in Relation to Age at Death. *American Journal of Human Biology* 3(4):369-375.
- Rice, G.H. 1983. *Changing Mortality Patterns in North Carolina, 1920-1972: A Regional Analysis*. PhD Dissertation. The University of North Carolina at Chapel Hill.
- Rickert, D. 1998. *Income Distribution and Health Outcomes: An Assessment of the Role of Poverty*. Ph.D. Dissertation. University of Alabama at Birmingham School of Public Health.
- Salomon, J.A. and Murray, C.J.L. 2002. The Epidemiologic Transition Revisited: Compositional Models for Causes of Death by Age and Sex. *Population and Development Review*, 28(2):205-228.
- Schofield, R. and Reher, D. 1991. "The Decline of Mortality in Europe", in *The Decline of Mortality in Europe*. Edited by R. Schofield, D. Reher and D. Bideau, pp. 1-17. Oxford, UK: Clarendon.
- Schofield, R., Reher, D. and Bideau (eds). 1991. *The Decline of Mortality in Europe*. Oxford, UK: Clarendon.
- Sharma, S., Malarcher, A.M., Giles, W.H., Myers, G. 2004. Racial, Ethnic and Socioeconomic Disparities in the Clustering of Cardiovascular Disease Risk Factors. *Ethnicity and Disease* 14(1):43-48.
- Shields, P.G., Lerman, C., Audrain, J., Bowman, E.D., Main, D., Boyd, N.R., and Caporaso, N.E. 1998. Dopamine D4 Receptors and the Risk of Cigarette Smoking in African-Americans and Caucasians. *Cancer Epidemiology, Biomarkers, and Prevention* 7(6):453-458.
- Starr, P. 1982. *The Social Transformation of American Medicine*. New York: Basic Books.
- Stewart, T.J., May, P.A., and Muneta, A. 1980. A Navajo Health Consumer Survey. *Medical Care* 18(2):1183-1195.
- Stull, D.D. 1972. Victims of Modernization: Accident Rates and Papago Indian Adjustment. *Human Organization* 31(2):227-240.
- Sundin, J. 1995. Culture, Class, and Infant Mortality during the Swedish Mortality Transition, 1750 – 1850. *Social Science History* 19(1):117-145.

- Svanborg-Eden K. and Levin B.R. 1990. "Infectious Disease and Natural Selection in Human Populations: A Critical Examination," in *Disease in Populations in Transition: Anthropological and Epidemiological Perspectives*. Edited by A. Swedlund and G. Armelagos G, pp. 31–46. New York: Bergin and Garvey.
- Sydenstricker, E. 1933. *Health and Environment*. New York: McGraw Hill.
- Thompson, W. 1929. Population. *American Journal of Sociology* 34:959-975.
- Trafzer, C.E. 1997. *Death Stalks the Yakama: Epidemiological Transitions and Mortality on the Yakama Indian Reservation, 1888-1964*. East Lansing, MI: Michigan State University Press.
- Trevino Garcia-Manzo, N., Escandon-Romero, C., Escobedo de la Pena, J., Hernandez-Ramos, J.M., Fierro-Hernandez, H. 1994. Amebiasis in the Epidemiologic Transition in Mexico: Its Morbidity and Mortality Trends in the Mexican Institute of Social Security. *Archives of Medical Research* 25(4):393-399.
- Trovato, F. 1988. Mortality Differentials in Canada, 1951-1971: French, British, and Indians. *Culture, Medicine and Psychiatry* 12:459-477.
- Veiller L. 1921. "Housing as a Factor in Health Progress in the Past Fifty Years," in *A Half Century of Public Health*. Edited by M. Ravenel, pp. 323–334. New York, NY: American Public Health Association.
- Wardlaw, G.M. and Insel, P.M. 1996. *Perspectives in Nutrition*, 3<sup>rd</sup> Edition. St. Louis: Mosby.
- Wilbur, R.L., Barnard, H.E., Glover, K., and Moses, W. 1931. *The White House Conference on Child Health and Protection, 1930; Addresses and Abstracts of Committee Reports*. New York: Century Company.
- Wilkinson, R.G. 1994. The Epidemiological Transition: From Material Scarcity to Social Disadvantage? *Daedalus* 123(4):61-77.
- Wilson, L.G. 1990. The Historical Decline of Tuberculosis in Europe and America: Its Causes and Significance. *Journal of the History of Medicine and Allied Sciences* 45:366-396.
- Winkleby, M.A., and Cubbin, C. 2003. Influence of Individual and Neighbourhood Socioeconomic Status on Mortality among Black, Mexican-American, and White Women and Men in the United States. *Journal of Epidemiology and Community Health* 57(6):444-452.
- Woods, R. 1991. "Public Health and Public Hygiene: The Urban Environment in the Late Nineteenth and Early Twentieth Centuries," in *The Decline of Mortality in Europe*. Edited by R. Schofield, D. Reher and D. Bideau, pp. 233-247. Oxford, UK: Clarendon.
- Young, T.K. 1994. *The Health of Native Americans: Toward a Biocultural Epidemiology*. Oxford: Oxford University Press.
- . 1996. "Recent Health Trends in the Native American Population," *Changing Numbers, Changing Needs: American Indian Demography and Public Health*. Edited by G. Sandefur and B. Cohen, pp. 53-75. Washington, D.C.: National Academy Press.
- \*Also published in *Population Research and Policy Review* 1997. 16:146-147.

## **Chapter 4: Theoretical Roles of Biology and Society in the Epidemiologic Transition among Native Americans**

### **Introduction**

High rates of disease among Native Americans have been a concern of researchers for decades. Infectious diseases had taken a tremendous toll on Native American populations in the historic past. More recently, disparities in chronic conditions have become a concern, prompting hypotheses that may explain the current disease burden. Anthropology has put forth the thrifty gene hypothesis as an explanation for obesity and associated pathologies among individuals and the New World syndrome to explain high rates of these pathologies among indigenous populations. These evolutionary suppositions have been popular explanations since the 1960s. However, do these biological models adequately account for the pattern of mortality observed among Native Americans? What other biological and non-biological hypotheses explain their mortality patterns? This chapter addresses these explanations and others and examines how well these concepts speak to the epidemiologic transition pattern of Native American populations such as the Prairie Band Potawatomi.

### **Biological Explanations**

#### **The Thrifty Gene Hypothesis**

Genetics has been implicated as a significant contributor to chronic conditions such as diabetes, obesity, and gall bladder disease (Shaffer 2005; Wendorf 1989). Neel presented a thrifty gene hypothesis to explain the rapid appearance of these chronic diseases in Native American populations (Neel 1962, 1982). This hypothesis suggests that genes that would have been advantageous during the hunter-gatherer past have become maladaptive in modern environments. Genes for storing fat as protection against times of food scarcity contribute to

obesity in a modern environment absent of starvation. Knowler et al. (1983) cited the thrifty gene hypothesis to explain the high rates of diabetes among the Pima (Akimel O'odham) Indians. The ability to store energy as fat in response to cycles of feast and famine would have been an adaptive advantage for the ancestral Pima. This ability is now seen as a maladaptation, because without periodic famines, the plentiful nature of food today leads to high rates of obesity, insulin resistance, and diabetes among the Pima.

Evolutionary pressures may have selected those Native Americans who could best conserve energy (Shaffer 2005). The origin of this thrifty genotype among Native American populations has been traced back to Paleoindian times. Supporters of the hypothesis suggest that some Paleoindian populations continued as hunter-gatherers in the subarctic regions of temperate North America spanning Wyoming to Arizona. Their subsistence practices featured dependency on big game species as the major food source, but during this time, the megafauna were going extinct and were becoming an unpredictable food resource. According to the hypothesis, Paleoindians who continued to rely on big game animals for food adapted to this dwindling resource by evolving a thrifty genotype that gave them a survival advantage during the famines that took place between successful big game hunts (Szathmary 1990; Weiss et al. 1989; Wendorf and Goldfine 1991). Those populations that moved south after the initial Paleoindian migration were under less selective pressure to develop a thrifty genotype. The gradual spreading of the ice-free corridor would have provided a larger subarctic environment for these later migrants and would have allowed them time to adapt to the smaller game and wild foods more common farther south. Thus, the Na-Dene (Athapaskan-speaking groups) were less likely to have developed the thrifty genotype, which is hypothesized to explain to some degree the variation

among Native American groups with regard to rates of diabetes and other obesity-related diseases (Wendorf 1989).

Caloric stress may not necessarily have been the principal environmental stressor, however. The arctic and subarctic environments of the Paleoindian period would not have produced a large variety of plant food resources. With animal protein as the primary food source, the Paleoindians would have experienced a low carbohydrate diet, although their caloric intake may have been adequate. Under such dietary conditions, selection would choose those individuals who could best utilize protein and fat as fuel. Thus, enhanced gluconeogenesis, the ability to free glycerol from triglycerides, and more efficient utilization of ketone bodies for energy would be favored (Ritenbaugh and Goodby 1989; Szathmary 1990; Young 1994). A recently published study of human coprolites from Ancestral Pueblo at Antelope Cave in Arizona suggests the original adaptation may not have been a response to periods of famine during big-game hunting, but to an extremely high-fiber diet. The low-fiber, high-glycemic index foods in modern diets thus may be implicated in the diabetes epidemic (Reinhard et al. 2012). Whatever the physiological mechanism may be, the thrifty gene hypothesis predicts that populations that suddenly experience a shift to a westernized diet and lifestyle will suffer from an increased rate of diabetes. Populations showing the highest rates of diabetes are precisely such populations (Wareharn 2004).

One of the fundamental assumptions of the thrifty gene hypothesis has been recently challenged. A basic assumption of the hypothesis is that hunter-gatherers experienced regular periods of famine. This assumption, however, has remained for the most part untested. Benyscheck and Watson (2006) compared the available food resources among ancient hunter-gatherers, contemporary hunter-gatherers, and farming populations and found no evidence to

assume that the foraging groups regularly experienced food shortages. Their analysis raises serious issues for the thrifty gene hypothesis. Archaeological, skeletal, and ethnographic evidence suggest that the early farming populations were more likely to suffer from periodic famines. Though the assumption of how and when a thrifty genotype evolved may be in question, the adaptation itself may still be relevant. The Pima, for instance, were a horticultural population that suffered from periodic drought and famine (Szathmary 1990).

Native Americans did experience a significant population decline that could have affected genetic susceptibility to disease. Recent research confirms that Native Americans underwent a genetic bottleneck following contact with Europeans. It has yet to be investigated fully how this bottleneck might contribute to the current epidemic of metabolic conditions. The authors did suggest that the genetic fitness of Native Americans did not appear to suffer any long-term damage or a substantial reduction in genetic diversity (O'Fallon and Fehren-Schmitz 2011).

### *Insulin Resistance*

Knowler et al. (1983) hypothesized that the thrifty genotype in Pima Indians operates through insulin resistance. Insulin resistance is a strong predictor of the development of diabetes among Pima Indians, as well as in Native Americans generally, and in the children of parents with diabetes (Rosenbloom et al. 1999). Insulin resistance is characterized by the failure of liver, muscle, and fat cells in the body to react normally in the presence of insulin; that is, to remove glucose from the blood stream (NDIC 2006; Shen, Reaven and Farquhar 1970). The condition was first described by Himsworth (1936), who noted that the blood-sugar levels of some people did not decrease significantly in the presence of insulin. In addition to elevated glucose levels, insulin resistance is associated with high blood pressure and dyslipidemia comprising elevated

triglyceride levels, a high percentage of small dense LDL molecules (Pattern B), and decreased HDL levels (Fox 2011).

Insulin is produced in the pancreas and secreted into the bloodstream. Its purpose is to induce absorption of glucose by cells from the blood for use as fuel. Insulin binds to protein tyrosine kinase receptors on the cell surface, signaling the cells to respond by increasing the number and action of glucose transport proteins in the plasma membrane. Insulin resistance occurs when the cells fail to respond appropriately. Obesity and physical inactivity have been cited as the most common contributors to insulin resistance. These conditions seem to be associated with a reduced number of cellular insulin receptors and with the failure of the receptors to trigger the tyrosine kinase response. Because insulin mediates fat and carbohydrate metabolism, as well as a number of other metabolic pathways, its effects in the body can be dramatic (Bloomgarden 1998).

Liver cells (hepatocytes) also help to regulate blood glucose levels. Glucose in the portal vein enters the hepatocytes, where insulin also stimulates the action of glycogen synthase and several other enzymes which transform glucose molecules into their stored form, glycogen. In this manner the liver absorbs more glucose from the blood than it releases, reducing blood glucose levels. When energy is needed, the process is reversed and glycogen in the liver is broken down and converted back into glucose. Among people with insulin resistance, this normal reduction of the production of glucose in the liver may not occur. Insulin resistance in the liver also acts to reduce the storage of glucose as glycogen (Fox 2011).

In liver and fat cells, insulin also promotes the synthesis of triglyceride from fatty acids in the bloodstream. Fatty acids can be used by many tissues as a source of fuel; when blood glucose levels drop, triglycerides are broken down to produce free fatty acids (lipolysis). Under



normal conditions, insulin, responding to elevated blood sugar levels, promotes the uptake of free fatty acids from the blood stream. In insulin-resistant fat cells there is reduced absorption of blood lipids and an increase in the hydrolysis of stored triglycerides, resulting in the elevation of free fatty acids in the blood (McGarry 2002).

Normal glucose metabolism is related to heightened insulin reactivity in skeletal muscle. This reaction is associated with an increase in the insulin regulating glucose transporters known as GLUT4, as well as in the enzymes that regulate phosphorylation, storage, and oxidation of glucose. Insulin promotes blood glucose absorption in insulin-sensitive peripheral tissues, and studies suggest that it can also stimulate vasodilatation in skeletal muscle to increase muscle blood flow in a dose-dependent fashion. An inability of insulin to stimulate muscle blood flow is a characteristic of insulin resistance syndrome among those who are obese or who have diabetes (Ivy 1997).

Insulin resistance is regularly found among people with a high fat concentration around the abdomen, a condition known as abdominal obesity. This is distinct from subcutaneous adiposity, or fat that is located between the skin and muscle wall, such as that found along the hips, thighs, and buttocks. Unlike subcutaneous fat, abdominal fat cells produce considerable amounts of chemical messengers called cytokines that are proinflammatory – that is they are capable of promoting inflammation. These proinflammatory cytokines may disturb normal insulin behavior in fat and muscle cells (Whitney and Rolfes 2011). Abdominal obesity is also related to nonalcoholic fatty liver disease (NAFLD), or increased adiposity in the liver. The condition results in elevated release of free fatty acids into the blood due to increased lipolysis (triglyceride catabolism) and an increase in gluconeogenesis by the liver. Each of these

intensifies insulin resistance and increases the likelihood of type 2 diabetes (Whitney and Rolfes 2011).

Insulin resistance syndrome, also sometimes referred to as Syndrome X or metabolic syndrome, is characterized by the comorbidities of insulin resistance, obesity, diabetes, hypertension, atherosclerosis, and dyslipidemia and is recognized as a significant contributor to reduced life expectancy. Associations with Alzheimer's disease and some cancers are also suspected. Excess consumption of fats and carbohydrates combined with a sedentary lifestyle are significant in the manifestation of the syndrome (Ten and Maclaren 2004). Insulin resistance contributes to high blood pressure, high cholesterol and diabetes, which in turn are risk factors for heart disease (Bloomgarden 1998). Thus insulin resistance has been indicated as the primary metabolic disorder leading to the other pathologies associated with Syndrome X (DeFronzo and Ferrannini 1991).

Studies of risk factors for heart disease and stroke among some Native American populations showed high rates of insulin resistance, renal failure, peripheral arterial disease, high blood pressure, high cholesterol, gallbladder disease, and diabetes (Casper et al. 2005; Shaffer 2005). The occurrence of multiple risks in Native American patients led to the conceptualization of "New World syndrome," in which these conditions among Native Americans were hypothesized to be genetic factors associated with lipid metabolism (Weiss, Ferrell, and Hanis 1984). The New World syndrome hypothesis is an application of Neel's thrifty gene hypothesis and the concept of metabolic syndrome as they apply specifically to Native American and other indigenous populations (Weiss 1990).

A refinement of the thrifty gene hypothesis is the "thrifty pleiotropic genotype" model, which explains the rise of chronic diseases late in life as the result of the effects of thrifty genes

with multiple effects that, while conferring a survival advantage early in life, become disadvantageous later in life. It is based on the original idea of Williams (1957) regarding senescence, and recognizes that the manifestation of chronic diseases in later life occurs after reproduction is nearly complete, thus there is little selective pressure against genes that contribute to such diseases. The model assumes that all organisms have evolved thrifty mechanisms for efficiently using nutrients, and that most alleles have multiple functions in different metabolic pathways. The rise of chronic diseases results from the recent overabundance of nutrients and the increase in life-span characteristic of modern societies (Crews and Gerber 1994; Gerber and Crews 1999).

### **Drifty Gene Hypothesis**

An alternative to Neel's hypothesis is the "drifty gene" hypothesis, which challenges the idea that natural selection is the evolutionary mechanism by which genes for obesity and diabetes arose. Speakman (2006, 2008) argues that famines were insufficiently selective to have a lasting impact on survivorship or reproduction; had there been such an evolutionary advantage for thrifty genes, they would have spread to all humans long ago. For individuals and subgroups to be carrying such genes today, they must have arisen through random mutations that were for much of human history selectively neutral. The frequencies for these genes thus drifted, inasmuch as they conferred neither selective advantage nor disadvantage. Only under modern circumstances are the genes and their products disadvantageous to health (Yilmaz et al. 2009). As a rebuttal to Speakman and in support of the thrifty gene hypothesis, the idea that famines and food shortages were common in the postagricultural era (Szathmary 1990) and a source for thrifty genes is suggested by Prentice, Henning, and Fulford (2008).

## **Thrifty Phenotype Hypothesis**

Another alternative to the thrifty gene hypothesis suggests that nutritional intake early in life may be a factor in the development of insulin resistance. Studies have directly related low birth weight to high BMI and abdominal obesity later in life (Oken and Gillman 2003). Poor nutrition *in utero* and as a neonate may alter the growth and operation of beta cells and insulin receptive cells. Insulin resistance may result later in life in association with obesity (Lappe 1994; Rosenbloom et al. 1999). A study of the Pima found prenatal malnutrition to be a strong predictor of diabetes in adulthood. The study was able to utilize birth weight as a measure of gestational nutritional stress (McCance et al. 1993). These findings were supported in a study of diabetes among the Havasupai, in which prenatal malnutrition again was found to be a predictor of diabetes in adulthood (Benysheck 2001). In addition to prenatal malnutrition, maternal gestational diabetes has been cited as a risk factor for diabetes in adulthood (Dabelea et al. 2000). A pattern of maternal malnutrition during gestation, high caloric intake in adulthood, and maternal gestational diabetes typified Native Americans in the 20th century, as they rapidly were transformed from a population that suffered recurring food scarcity to one consuming a high kcal, high-fat, high-carbohydrate diet (Benysheck 2001).

These studies suggested an alternative hypothesis to the population genetic explanation of diabetes, sometimes referred to as the thrifty phenotype hypothesis or Barker hypothesis. This hypothesis proposes that the observed relationship between poor fetal and infant development and diabetes mellitus later in life is the result of inadequate nutritional intake early in life. This nutritional inadequacy causes lasting changes in glucose metabolism, including a reduction in insulin production as well as cellular insulin resistance (Barker 1994, 1997; Hales and Barker

1992; 2001). More recent studies provide more evidence for this effect but also point to fetal *over* nutrition as a possible cause of diabetes later in life. Speculation remains regarding the mechanisms leading to the observed effects in adulthood (Kanaka-Gantenbein 2010). To complicate things further, other evidence suggests a genetic connection between prenatal and early postnatal growth and chronic diseases later in life. Demerath et al. (2004) suggest that telomere attrition may underlie the fetal origins of adult diseases described by the thrifty phenotype hypothesis.

## **Epigenetics**

Continued research into the thrifty genotype and thrifty phenotype hypotheses has led to a new hypothesis that attempts to correct for their limitations, called the thrifty epigenotype hypothesis. This hypothesis suggests epigenetic effects are the links between early pre- and postnatal environmental events and the later manifestations of disease in adulthood (Stöger 2008). Epigenetics is the relatively new study of changes in gene expression that are caused by factors other than changes in DNA sequence. These changes may be due to such things as DNA methylation or histone modification, which are mechanisms that affect gene expression without altering the sequence of the actual genes. These changes in gene expression can be maintained through the process of cell division throughout the cell's life and can also persist over many generations (Bird 2007). The process is seen as adaptive, because the early life environment provides clues to future environmental conditions, thus allowing offspring to adapt their biological development trajectory. Whereas genetic adaptation through natural selection proceeds slowly in a population, epigenetic adaptation increases biological plasticity in an organism's lifetime, allowing a more immediate adjustment to environmental stress (Gluckman et al. 2008; Gluckman, Hanson, and Spencer 2005; Kuzawa and Thayer 2011).

The thrifty epigenotype hypothesis posits that inasmuch as energy metabolism is a highly conserved mechanism in all species, all humans have thrifty genes. These metabolism genes are complex, buffered against allelic heterogeneity and thus, canalized and resistant to mutational changes. Genetic polymorphisms play a minor role in disease susceptibility, whereas epigenetic variation predominantly determines disease expression; epigenetic changes are heritable. Leptin has been identified as a good candidate for the acquisition of these thrifty epigenetic traits because its genes encode a hormone regulating appetite and energy metabolism. Among the Pima Indians, blood leptin concentrations are lower in individuals with a tendency to gain weight. Epigenetic variation in the promoter region of the leptin gene (LEP), which is methylated in human body tissues, may explain low plasma concentrations of this adipose tissue hormone. LEP may be responsive to environmental circumstances and conditioned to acquire a thrifty epigenotype. Allelic LEP variants have yet to be identified, and it appears that LEP mutations and allelic variation associated with obesity are rare in humans (Stöger 2008).

Epigenes have been implicated in racial health disparities. Minorities suffer not only from a disproportionately high burden of cardiovascular diseases compared to whites, but also from higher rates of the pre- and postnatal health conditions now held to be the precursors of these chronic illnesses. An environmental and social origin of premature births and low birth weight among minorities reflects such conditions as poor prenatal care and effects of maternal stress due to discrimination. Considering the correlation between birth weight and cardiovascular diseases in adulthood, epigenetic mechanisms provide the links between prenatal environmental factors and adult racial health disparities in chronic diseases such as diabetes, hypertension, strokes, and coronary heart disease. Socially constructed and imposed racial identities and surrounding circumstances influence the developmental process and manifest as

biological configurations that negatively affect health and can be passed down to subsequent generations, maintaining health disparities (Kuzawa and Sweet 2009).

## **Summary**

Ever since Neel's first elucidation of the thrifty gene hypothesis in 1962, numerous attempts have been made to confirm or refute the hypothesis. Archaeological and ethnographic evidence call into question the initial explanation of cycles of feast and famine during the Paleolithic as the cause of thrifty genes. New knowledge and technologies in the field of genetics indicate that the situation is more biologically complex than originally thought. The traits that characterize the New World syndrome are complex traits, making it unlikely that a simple gene or set of genes can be responsible for the observed phenotype. Any biologically based explanation will have to account for the interaction between genes and the environment.

## **Societal Explanations**

### **Structural Violence**

Structural violence explains the higher rates of morbidity and mortality experienced by those at the lower end of the socioeconomic spectrum, compared with those who occupy a higher socioeconomic rank (Gilligan 1996). Historical legacies and economic processes work to limit an individual's ability to flourish. As a concept, structural violence prompts the examination of the social mechanisms of oppression. Structural violence thus occurs when low socioeconomic status prevents some groups from accessing the benefits of technological and economic progress. It compromises access to health care in that medical services exist as a commodity with costs, and as such are available only to those who can pay for them (Farmer 2004; Farmer et al. 2006). Structural violence has operated against Native Americans primarily through federal Indian

policy. Whether through malicious or benevolent intentions, the system has negatively affected Native American health.

### **Basis of Federal Indian Policy**

In what manner did federal Indian policies shape the pattern of epidemiologic transition and current health conditions of Indian people? In general it has been recognized that federal Indian policies have had a dramatic effect on Indian sovereignty and ability to flourish physically, culturally, and economically. Indian health and wellbeing have often been at the cost of these policies, which featured the only federal bureau devoted to all aspects of the lives of a specific group of people, until the Indian Health Service was created under the U.S. Public Health Service in 1955. In this all-encompassing bureaucracy, policies toward sovereignty, tribal structure, social structure, education, and, of course, health services all affected the actual health conditions of the people. As a result, Indian health has been affected by decisions made by government legislators and administrators.

The relationship between Indian tribes and the U.S. government is founded in the Constitution:

Article I, Section 8 –

The Congress shall have Power...to regulate Commerce with foreign Nations, and among the several States, and with the Indian Tribes;

Article II, Section 2 –

[The President] shall have Power, by and with the Advice and Consent of the Senate, to make Treaties, provided two thirds of the Senators present concur.

The commerce clause of the U.S. Constitution gives the federal government the power to regulate relationships with the Indian tribes (Stull 1984; Tyler 1973). The treaty clause of the



U.S. Constitution gives the federal government the power to negotiate treaties on behalf of the nation. Through these two clauses, which together form the basis of federal Indian policy, the U.S. government has established a singular relationship with Indian tribes: the tribes are viewed as domestic dependent nations within the U.S., and as a result the federal government takes on a trust responsibility to the Indians based on legal precedence, treaties, and congressional legislation. This trust relationship obligates the federal government to significant legal and moral responsibilities (IHS 2005). These sections of the Constitution provide the operational framework by which structural violence operates against Native Americans.

A specific set of principles guide federal Indian policy. These are tribal sovereignty and property rights; federal power, obligations, and jurisdiction; and reserved rights:

1) *Tribal Sovereignty and Property Rights*. Indian tribes were initially recognized as having external sovereignty, in which they were viewed in the same manner as independent nations. This external sovereignty has been eroded over the years, though the government still acknowledges tribal internal sovereignty, or the right of tribes to be self-governing (Getches, Wilkinson, and Williams 2004; Stull 1984).

2) *Federal Power, Obligations, and Jurisdiction*. The principle of federal power and obligations has as its source the commerce clause of the Constitution, by which the U.S. government averted wars with and among the Indians; the government also attempted to inhibit scams against the Indians through commerce regulation (Getches, Wilkinson, and Williams 2004; Stull 1984). Federal laws and treaties thus shape a distinctive association between Indians and the government, establishing the principle of federal power and obligations. This distinctive association exists in the form of federal authority, as opposed to state authority, regarding Indian legal issues. It provides the federal

government with jurisdiction over Indian affairs and also obligates the federal government to protect and serve certain Indian interests. Congress has plenary power over Indian affairs. That is, the Congress may pass legislation without tribal agreement or prior approval, although this power of Congress is not technically absolute. The U.S. Supreme Court can review plenary legislation to ensure that it is consistent with congressional obligations to Indians. However, no such congressional legislation has ever been rejected by the Supreme Court.

3) *Reserved Rights*. The principle of reserved rights dictates that except in cases where tribes have signed away their rights or have had their rights taken through congressional plenary power, the tribes preserve all assumed rights. Any right not specifically defined by a treaty or law is considered to be reserved by the tribes. Indian water and mineral rights have been preserved by this principle. The principle of reserved rights also requires a conservative interpretation of treaties and laws and requires that interpretations be consistent with Indian understanding of the accord at the time it was made (Getches, Wilkinson, and Williams 2004; Stull 1984).

The legal status of the trust relationship was delineated in the 1823 Supreme Court case *Johnson v. McIntosh*, which stated that Indian tribes had a legal title to the lands they occupied. However, that legal title was not absolute, in that Indian tribes could only transfer that title to the U.S. government (Stull 1984). Chief Justice John Marshall cited European rights of discovery as conferring first upon the crown and subsequently upon the U.S. government, absolute title (Deloria 1985; Getches, Wilkinson, and Williams 2004).

Despite the deceitful manner in which many Indian treaties were written, they still form the legal basis for Indian and U.S. government relations. Congress could not simply ignore the

treaties made in the past, and affirmation of the validity of those treaties was also written into the 1871 Indian Appropriations Act. As the courts recognized the misleading circumstances under which treaties had been negotiated and the trust obligation of the government toward the tribes, they employed canons of construction for interpreting treaties as a way of righting past injustices. These are a set of conventions used by the courts to interpret treaties in a manner to the advantage of the Indians. These conventions state that vague language is to be settled preferentially for the good of the tribes; that treaties are to be read as they were comprehended by the tribal signatories; and that treaties are to be generously construed in support of Indian interests (Prucha 1997; Stull 1984).

A large number of the treaties listed health care as a part of federal compensation for Indian territories (IHS 2005; Kane and Kane 1972). By the end of the treaty era in the early 1870s, at least 24 treaties guaranteed some form of health services (Campbell 1994). Therefore, Indian health is closely tied to federal Indian policies, but actual federal involvement in Indian health was limited during much of the 19th century. The earliest efforts lacked funding and consistency (Kane and Kane 1972), although as early as 1802 Indians in the vicinity of military forts were given periodic care by military physicians. Health services for Indian people were formally established in the U.S. Department of War in 1824, when vaccination of Indians was viewed as a public health measure and was an incentive to provide some services to Indians. The U.S. Congress in 1832 appropriated \$12,000 to provide smallpox immunizations to Indians, although this was more likely a measure to protect soldiers at U.S. forts than to save Indian lives. In 1836 the U.S. Government began to provide health care services and medical personnel to the Ottawa and Chippewa in exchange for their promise to remain on their reservation. Similar arrangements were made in treaties with other tribes (Bergman et al. 1999; Brophy and Aberle

1966; IHS 2005; Kane and Kane 1972; Rhoades, Reyes and Buzzard 1987). Control of medical services for Indians shifted from the War Department to the Department of the Interior when the Office of Indian Affairs (OIA) changed departments in 1849, but the first OIA health programs were not developed until 1908. In 1909 a medical supervisor was first appointed by the bureau. Funding specific to Indian health totaling \$40,000 was first appropriated in 1911 (Bergman et al. 1999; IHS 2005; Tyler 1973).

## **Assimilation**

Movements to reform the government's treatment of Indians first in the 1860s from non-Indians who lived closely with the natives, and were upset by their poor treatment and fraudulent management of Indian affairs. Most of these early reformers had a genuine concern for the Indians, inspired by Christian humanitarian beliefs. This movement would eventually lead to a new Indian policy that would dominate U.S.-Indian relations into the next century (Fritz 1963). The OIA programs and funding mentioned above and greater federal involvement in Indian health came about during the progressive era, a period of reform that brought about the government's assimilation policy in Indian affairs. The latter years of the 19th century had signaled the beginning of the assimilation policy, which was a concerted effort to exterminate Indian culture and integrate Indian people into the greater American society (Stull 1984). Assimilation policy grew out of progressive era thinking that assimilation would eventually bring Indian people up to the same level of civilization as the rest of America (Barsh 1991; Fiorentino 1999). Much of 20th century Indian policy, particularly in the early years of the century, featured legislation based on assimilation policy. The government focused on individual Indian people as opposed to Indian tribes in implementing the Dawes Act and boarding schools (Prucha

1984; Tyler 1973). The first half of the 20th century also resulted in greater governmental responsibility for Indian health (Garrett 1994).

Reformers of Indian policy concluded that tribal kinship systems and communalism prevented assimilation by undermining individualism, so they turned to federal Indian policy to break up extended tribal families into male-dominated nuclear families, modeled after the U.S. middle-class households envisioned in the Homestead Acts. The reformers advocated for subdivision of tribal lands into individual allotments and the dismantling of tribal governments. They believed that allotment would promote individualism and economic independence, overcome and destroy tribalism, and encourage Indian adoption of the values and culture of the rest of America (Stremlau 2005). Various groups viewed allotment as a solution to the Indian problem: humanitarians, Indian sympathizers, experts on Indian affairs, and “land-grabbers,” all of whom considered allotment not only desirable but also inevitable. Much of the contemporary debate concerned whether allotment should be voluntary or compulsory (Washburn 1986).

The plenary power of Congress was affirmed in the U.S. Supreme Court ruling in *United States v. Kagama* [118 U.S. 375 (1866)]. This decision provided for governmental intrusion into tribal affairs under the guise of “protection”. The ruling gave the government the authority to impose assimilation efforts on Native Americans (Wilkins 1997). The Dawes Act combined the interests of speculators, reformers, humanitarians, intellectuals, and settlers. Indian interests, however, were mostly inconsequential. It was generally recognized that Indians would lose a significant amount of land. But within the context of the anticipated positive aspects of assimilation and equality, this reduction in the land base was seen as acceptable. Those who viewed allotment as a positive change for Indians failed to anticipate that the Indians would lose not only their surplus lands but their individual allotments as well. Indian people had not been

adequately prepared for the changes in subsistence and their way of life. Furthermore, in the name of assimilation, the government was coercing the Indians into adopting a way of life that was fading into history. Agribusiness was developing across the U.S. at the time, and many Americans were leaving their farms and moving to the growing cities, unable to compete with the rise of corporate and mechanized farming. The Dawes Act and subsequent amendments would represent official government Indian policy until 1934, despite early signs that severalty was disenfranchising Indians of their land (Clifton 1998; Washburn 1986).

Additional efforts to assimilate Native American populations involved educating children in boarding schools that suppressed all things Indian and taught the youngsters how to live as whites. "Kill the Indian to save the man" was the philosophy of assimilation (Pratt 1973). This meant that Indian culture was to be replaced by white culture, and the replacement would be complete. Not only was a new subsistence method to be forced upon the Native Americans, they were expected to adopt the social arrangements, religion, behavior, and attitudes of the dominant U.S. society (Prucha 1976). Indian education at boarding schools also grew out of assimilation policy. Policy makers viewed education as the tool to transform Indian children into civilized people (Grinde 2004), but the boarding schools also served to aggravate and spread diseases among their residents. Inadequate funding, poor treatment of students, crowded living quarters, and nutritionally deficient foods were all implicated in disease outbreaks among the students. Such diseases then spread to the reservation communities when sick children were sent home (Adams 1995; Putney 1980). High disease rates at boarding schools prompted OIA officials to assign doctors or nurses to the schools. Despite these measures, many Indian children died at the schools (Adams 1995; Joe 1991). Tuberculosis was responsible for over 25% of all Indian deaths, and the yearly incidence was increasing. Health officials recognized the importance of

diet in the prevention of tuberculosis, but the Office of Indian Affairs cut funding for rations among boarding school children, and the rates of TB subsequently increased. Even when funding at boarding schools was restored, the nutritional needs of those living on the reservations were virtually ignored. Nutrition was also cited as a concern with trachoma, which, although not fatal, was a major cause of disability and blindness among Native Americans (Meriam et al. 1928); in this case, however, the emphasis on nutrition ignored the fact that trachoma was highly infectious and required a different approach (Putney 1980).

Though committed to Indian health, the Indian Affairs commissioners during the early 20th century were heavily influenced by progressive-era policies. These paternalistic policies sought little input from the reservation communities themselves. Commissioner Cato Sells viewed the adoption of farming as a way to elevate Indian health by eliminating poverty, idleness, and improving nutrition (Putney 1980). A “New Declaration of Policy” was announced by Commissioner Sells in April 1917. This policy favored separate government responsibilities for mixed-blood and full-blood Indians. “Competent” or “able-bodied Indians” were to be given full and individual control of their property (Tyler 1973).

After the close of the progressive era, Congress passed the Snyder Act in 1921 (P.L. 67-85; 42 Stat. 208), which stipulated continuing authority of the federal government over Indian programs. The Snyder Act states:

Sec. 13. [25 U.S.C. 13] The Bureau of Indian Affairs, under the supervision of the Secretary of the Interior, shall direct, supervise, and expend such moneys as Congress may from time to time appropriate, for the benefit, care, and assistance of the Indians throughout the United States.

Among the priorities listed for funding was a specific reference to Indian health. The Snyder Act included the phrase “For relief of distress and conservation of health,” and thus provided the authority for the federal government to provide health care services to American Indian Tribes (IHS 2005). The Indian Citizenship Act of 1924 granted U.S. citizenship to American Indians. The act was signed into law by President Calvin Coolidge on June 2. Many Indians had already become citizens through allotment, so the act was primarily a response to the large number of Indians who volunteered to serve during World War I. The act was also a part of the assimilation policy and was another attempt to merge Indian people with the greater U.S. population (Stull 1984; Tyler 1973).

Indian health status continued to be substandard despite the Snyder Act and the Indian Citizenship Act, and over a quarter of a century of efforts by the Office of Indian Affairs failed to control diseases among the Indians, among whom the death rate was significantly higher than for other racial groups in the U.S. (Guthrie 1929). Having gradually become more responsible for Indian health, the OIA was compelled by the poor health status of Indian people to establish a special health division in 1924 (Kane and Kane 1972). The situation prompted Secretary of the Interior Hubert Work to encourage OIA and United States Public Health Service (USPHS) cooperation and to commission a comprehensive study of Indian health status. In 1926 public health experts were assigned to the OIA from the USPHS, which signaled the beginning of the participation of the USPHS Commissioned Corps in Indian health care efforts. A separate study on Indian health by the Institute for Government Research (1928), known as the Meriam Report, ridiculed the results of the policies of the progressive era (IHS 2005; Putney 1980). The Meriam Report indicted allotment policy of the Dawes Act in the poor conditions of the Indian people (Meriam et al. 1928; Stull 1984). The report concluded that allotment did not include adequate



education on the use and value of land, and thus largely failed to accomplish its objectives. Allotment resulted in a great loss of property and a substantial increase of government administration of Indian affairs without producing the economic improvement of Indian people (Meriam et al. 1928). The government's promotion of the policy that individual land ownership would serve to civilize the Indians actually produced the opposite outcome. Land ownership often allowed Indians to sell their allotment for an income that was only temporary and eventually left them penniless (Meriam et al. 1928). Allotment failed to make farmers out of Indians, and it failed to assimilate and "civilize" them (Tyler 1973).

The Meriam Report recommended more and better doctors, nurses and clinics, as well as more disease-prevention efforts. Additionally, the report called for better statistics and cooperation with state and local health entities. The Meriam Report also identified education as a major prerequisite for improving Indian health. Efforts were needed to convince Indians that doctors and nurses could actually improve their health. The changes recommended by the Meriam Report would not be implemented until the 1930s and 1940s (Meriam et al. 1928; Tyler 1973), although the Enforcement of State Laws affecting Health and Education Act of February 15, 1929 (45 Stat. 1185) empowered state health officials and agents to enter Indian reservations to make systematic evaluations of the health, sanitation, and educational conditions on Indian lands.

President Roosevelt's New Deal included the Indian Reorganization Act (IRA). The Wheeler-Howard Act of 1934 ended allotment and preserved the trust status of Indian land; restored to tribes unsold surplus lands created by the Dawes Act; provided resources for the preservation and management of Indian agricultural livestock and lands; extended loans to tribes for economic enterprises; established Indian preference for hiring workers in the Office of Indian

Affairs; gave tribes the opportunity to establish governments to administer their affairs; and gave tribes the option of rejecting the IRA if they wished. The opportunity to reject the IRA was a major shift in the government's stance regarding Indian policy, but a downside of this option was that tribes were given only one chance to accept or reject the act (Stull 1984). Critics of the IRA suggest that the legislation encumbered participating Indian tribes with a Western governmental and economic system unfamiliar to them (Bee 1999; Prucha 1984). Other scholars suggest that without the IRA, tribal government in any form would have long ago ceased to exist (Washburn 1984). Out of the Indian New Deal also came the Johnson-O'Malley Act in 1934, which encouraged federal cooperation with states and territories to improve Indian education, health, and general welfare. It provided funds to states for these purposes, thus improving OIA delivery of services as well as compensating states for services they claimed they were already providing (Tyler 1973).

## **Termination**

After the Second World War, the governmental policies that shifted toward termination coincided with rapid social changes among Indians. As social and economic services were eliminated on reservations, many Indian people entered the cash economy, left the reservations, moved to more urban areas, and began to consume more manufactured goods. The shift of health care responsibilities from the Bureau of Indian Affairs (the Office of Indian Affairs was changed to the BIA in 1947) to the United States Public Health Service indicated not only an administrative restructuring, but also the rise of a new medical philosophy. The poor health of American Indians was no longer attributed to an uncivilized existence, but to a lack of sufficient health education. Significant health changes took place as the Indian way of life and political relationship with the government changed.

In 1948 the health of Indian people was still inadequate, according to the findings of the Hoover Commission and the American Medical Association: the rate of infant mortality and the number of deaths due to infectious diseases were still unacceptably high (IHS 2005). With such health disparities in mind, the Hoover Commission viewed assimilation as a primary goal for alleviating Indian economic and health disparities, thus laying the groundwork for termination policy (Deloria 1985; Tyler 1973), which became the prevailing policy of the federal government in the 1950s (Prucha 1984). With termination the U.S. Congress planned to end the special relationship of the federal government with Indian tribes. Indian people were to be given citizenship, with all its rights, responsibilities, and privileges, in order to reduce Indian dependence on the government. Legally, termination negated federal trusteeship over Indian affairs and reservations, and the government's recognition of tribal sovereignty. Tribes and tribal members would no longer be excluded from the jurisdiction of state laws (Getches, Wilkinson, and Williams 2004). Following the election of Republican Dwight Eisenhower in 1952, those favoring termination pushed hard for the policy (Burt 1986), which was implemented in House Concurrent Resolution (HCR) 108 in 1953 (Prucha 2000; Stull 1984).

Urbanization was a major feature of the Indian experience during the 20th century (Prucha 1984). As termination became the policy of Congress, the Bureau of Indian Affairs began encouraging Indians to relocate in urban areas where employment would be more widely available. Additionally relocation was touted as a cost-effective alternative to dealing with poverty and other problems on Indian reservations (Burt 1986). Tribal members complained that the relocation policy was termination at the level of the individual. Indeed, relocation took people from their traditional influences and hastened the pace of assimilation by immersing Indians into the broader American culture (Stull 1984). Termination policy in the post-war years

also led to the modernization of many Indian people, accompanied by shifts in Indian morbidity and mortality patterns (Tyroler and Patrick 1972). Criticism of termination came quickly.

Numerous tribes and Indian organizations recognized the effects termination would have on Indian tribes, lands, and culture (Caldwell 1956). Termination more often led to the deprivation and impoverishment of the Indians than to their independence (Walch 1983). Moreover, closure of Indian health facilities mirrored termination of Indian tribes (Campbell 1994).

### *Indian Health Service*

Some policy makers maintained that the aims of federal Indian policy in the 1950s—termination of special status and assimilation—could not be achieved so long as Indian health status was so much worse than that of the rest of America (Kunitz 2004). Based on the studies and reports indicating the deficient nature of Indian health and health care, the responsibility of Indian health was transferred from the BIA to the USPHS in the Department of Health, Education, and Welfare in 1955 (IHS 2005). While some lawmakers who supported the Transfer Act saw it as a way to improve Indian health, others saw it simply as a part of termination—an attempt to end the special legal relationship between Indians and the government by assimilating Indian people to the dominant American culture (Bergman et al. 1999). Once the health of Indians was sufficiently improved, the government would cease to provide free health services, and Indians would be treated in the same manner as all other American citizens (Kunitz 2004).

With the Transfer Act, approved by President Eisenhower in 1954 (PL 83-568), erstwhile responsibilities of the Secretary of the Interior relating to the health of Indian peoples shifted to the Surgeon General of the USPHS. On July 1, 1955, the health program personnel, hospitals, health centers, infirmaries, and other health-related entities of the BIA were placed under the authority of the newly established Indian Health Service (IHS) (Bergman et al. 1999; IHS 2005;

Tyler 1973). In 1956, the Congressional Committee on Appropriations of the House of Representatives instructed the USPHS to conduct a broad survey of Indian health conditions (IHS 2005). The results of the survey were submitted to Congress in 1957. “Health Services for American Indians” (Perrott and West, 1957), commonly referred to as the “1957 IHS Gold Book” due to its gold cover, concluded that: a significant federal Indian health effort was necessary; community health programs and resources should be developed in cooperation with each individual Indian society; federal programs should be planned in each Indian community; state and local services should be made available to Indians; and obligations and responsibilities to Indian people should be made on a nondiscriminatory basis. The 1957 IHS Gold Book hinted at greater Indian self-determination and suggested a greater federal commitment to Indian welfare, counter to the government policy of termination.

As trachoma and tuberculosis among Indian people were brought under control in the 1940s and 1950s, chronic diseases emerged to take their place. Heart diseases, substance abuse, diabetes, cancer, accidents, and violence all rose in prevalence. To address these emerging health problems, the federal government eventually passed legislation in the 1970s aimed at providing Native Americans with greater control of federal and health care resources (Campbell 1989; Kelly 1952; Tyler 1973).

### **Self-Determination**

Termination did not produce the predicted outcomes, and was harshly criticized by lawmakers and Indians. Specifically targeted for termination by HR 4985 in 1954, the Prairie Band Potawatomi resisted termination in an attempt to maintain their tribal system (Mitchell 1995). Finally listening to the outcry from tribes, some policy makers began to recognize the importance of Indian culture and began to work for greater Indian control of government

resources (Caldwell 1956; Josephy, Nagel, and Johnson 1999; Kennedy et al. 1969; Stull 1984). Democrats reclaimed the White House and gained congressional seats in 1960. Federal Indian policy soon shifted away from termination toward a renewal of federal support for tribal communities (Burt 1986). In 1961 the Commission on the Rights, Liberties, and Responsibilities of the American Indian found persistent health disparities between Indians and the rest of the U.S. population. The commission related the poor health status of Indians to their poor economic conditions and made recommendations that included improving Indian socioeconomic status and education (Brophy and Aberle 1966; Campbell 1994). The last termination took place in 1966 (IHS 2005). That same year, President Johnson rejected termination in favor of self-determination in a speech before congress, and Senator George McGovern introduced a resolution in favor of greater self-determination in Indian affairs (Johnson, Champagne, and Nagel 1997).

In 1965 the Office of Economic Opportunity (OEO) funded the Community Health Representative (CHR) Program at the Pine Ridge Reservation in South Dakota. The program is seen as the first move toward tribal management of Indian health affairs. Congress appropriated more funds for the CHR program to the IHS in 1968 (Bergman et al. 1999). Prior to this time, programs for Indians had been managed by outsiders, largely without Indian input or participation (Bergman et al. 1999). Senate Concurrent Resolution (SCR) 11 in 1968, while harshly criticizing termination and recognizing the importance of Indian culture and the need for greater self-determination, also argued for better health care, education, economic development, and trust protection (Stull 1984). Public Health Service control of Indian health programs was criticized for not producing a healthier Indian populace and for its disinterest in providing preventive care or in identifying the underlying causes of Indian health disparities. Instead,

USPHS chose to focus on temporary cures and treatment as opposed to addressing the underlying social, environmental, and nutritional causes of disease. USPHS was accused furthermore of denying Indians any voice in determining health priorities or designing health programs (Cahn 1970).

President Richard Nixon's Indian Policy Statement in 1970 had the most dramatic impact on Indian relations with the U.S. government since the conclusion of the treaty era (IHS 2005). Nixon observed that government relations with Indians were grounded in the U.S. Constitution and in subsequent treaties between the government and the Indian tribes. Thus, a government-to-government relationship existed between the United States and the various tribes. Nixon advocated the concept of tribal self-determination, suggesting that federal programs for tribes be assumed and managed by the tribal governments themselves. Congress affirmed President Nixon's Indian policy statement by passing the Indian Self-Determination and Education Assistance Act (ISDEAA) (Public Law 93-638) in 1975. The law stipulates that an Indian tribal government may ask to assume the function of any BIA or IHS program (IHS 2005). The ISDEAA was the most significant law regarding Indian policy since the 1934 Indian Reorganization Act and was a key change from the termination policy of the previous two decades (Stull 1984; Stull, Schultz, and Cadue 1986).

The most significant legislation affecting Indian health was the Indian Health Care Improvement Act (IHCIA) of 1976. Prior to the IHCIA the only statute sanctioning health care services for Indian people was the 1921 Snyder Act. The Snyder Act was sufficiently broad to allow the IHS to provide health care services; however, the ambiguous focus of the act inhibited long-range development and destined Indian health programs to the uncertainty of the annual budget allocation process. Thus, rationed services were standard for the IHS. Should the

funding budgeted for the year be used up before the year ended, no more money would be forthcoming and services would be restricted (Bergman et al. 1999). With the IHCIA, Congress recognized that federal services for Indian health are a part of the government's special legal relationship with American Indians and thus a responsibility of the government. The goal of the government should be to provide the necessary health services to elevate the health status of Indian people and to encourage tribes to take an active role in the development and administration of services. Congress stipulated that the IHCIA funding was meant to augment rather than replace standard IHS funding (Bergman et al. 1999).

Congress identified two primary national goals in passing the Indian Health Care Improvement Act in 1976: 1) to elevate the health status of Indian people to the highest possible level; and 2) to attain the greatest level of participation of Indian people in health programs. Other new initiatives included the development of a scholarship program to support Indian education, construction of Indian health facilities, the provision of sanitation services for Indian reservations and homes, and the extension of Medicare and Medicaid reimbursements for services provided to eligible Indians. Congress mandated that in addition to simply providing health care to Indians, the IHS must also eliminate health disparities between Indians and the rest of the U.S. population (IHS 2005). The Indian Health Care Improvement Act solicited tribal involvement by requiring that each tribe list its community's particular health care requirements and develop a broad strategy to address those requirements. More than 90 percent of the eligible tribes participated in strategy development, and their recommendations became the foundation of the renewal of the Indian Health Care Improvement Act in 1980 (Bergman et al. 1999).

Federal Indian policy following the Indian Self-Determination Act focused on tribal sovereignty, economic self-sufficiency, and cultural self-determination. In the many cases in



which a sustainable and healthy reservation-based economy was absent, the government supplied funding. Transfer payments and government employment provided the appearance of economic self-sufficiency and self-determination to reservations (McGuire 1990). This legislation and funding of the 1970s held promise for Indian people to improve their lives. However, the federal money that poured into Indian reservations during the 1970s failed to produce significant economic development by the 1980s (Prucha 1984; Stull 1990). The financial base had been arbitrarily set by government bureaucrats with little regard for program effectiveness, workload, or local needs (Kane and Kane 1972). By 1980, federal funding provided employment for over half of all jobs on the reservations. Reservation bureaucracies benefited, but to pacify federal granting agencies and secure more funding, tribal programs often forfeited usefulness, sustainability, and culturally suitable approaches. With the coming of the Reagan administration, reservation economies suffered from significant reductions in funding (Barsh and Diaz-Knauf 1984). Subsequently, the economic progress made by tribes following the Indian Self-Determination Act was soon eliminated by federal budget cuts in the 1980s (Stull 1990; Stull, Schultz, and Cadue 1986). Thus, whereas the ISDEAA allowed tribes to assume control of tribal health services from the IHS and provided tribes more freedom to design and administer programs, funding for such programs was vulnerable to political policies and changing priorities with each new administration (Joe 1991; Smith-Morris 2004).

Self-determination efforts prior to 1988 were criticized as suffering from procedural, fiscal, and fundamental problems. Procedurally, IHS and BIA have been criticized as being overly bureaucratic and unresponsive to tribal requests for information. Fiscally, in addition to the problems mentioned above, funding provided to tribes for programs reflected program costs of the federal agencies, not the actual costs for tribal implementation. Though IHS funding

doubled between 1972 and 1985, so did the IHS eligible population, actually leading to a reduction in funding per eligible recipient after adjusting for inflation. Fundamentally, the IHS and BIA continued to be the primary operating authorities by identifying problems, designing programs, and defining the criteria for success, with little input from tribes (Stuart 1990; U.S. Congress 1986).

Governmental control of economic and political resources perpetuated welfare dependency (Joe 1991). Tribal dependence on federal funding and oversight hindered self-determination. Despite the Indian Self-Determination Act of 1975 and the flow of federal monies into reservation communities, Indian people on reservations continued to be the poorest group in the U.S. (Darian-Smith 2004). Tribes viewed gaming as a way to generate revenue and escape dependence upon unreliable federal funding and authority. High-stakes Indian gaming had begun as early as 1979, when the Seminole Tribe of Florida established a bingo parlor. Other tribes followed suit, including the Kickapoo and Potawatomi, claiming tribal sovereignty allowed them to operate gaming facilities without state or local government approval. The Indian Gaming Regulatory Act (IGRA) was enacted in 1988 to establish state and federal jurisdiction of gaming on Indian lands (Darian-Smith 2004).

The Indian Self-Determination Amendments of 1988 (PL 100-472, Title III of ISDEAA) first allowed tribes to contract with the BIA to assume the control of programs on a trial basis. After passage of the Indian Health Care Amendments of 1988 (PL 100-713), tribes and urban Indian organizations more often began to manage their own health programs, and IHS responsibilities for directly providing health care services decreased (Bergman et al. 1999; Berry et al. 2004). Also in 1988, the IHS was elevated from bureau to agency status within the USPHS (52 FR 47053), granting the IHS Director greater authority (Bergman et al. 1999; IHS 2005).

IHS elevation was designed to provide IHS leaders with better access to policy makers at the Department of Health and Human Services and was viewed as a way to involve other USPHS agencies in researching and solving the primary health problems experienced by tribes (Joe 1991).

The tribal contracts established by the 1988 demonstration project were expanded to 30 tribes with the passage of the Tribal Self-Governance Demonstration Project Act of 1991 (PL 102-184). Titles I and V of the ISDEAA permitted tribes to assume responsibility for health care services, allowing them to contract with the IHS through self-determination contracts under Title I or compact with the IHS through self-governance compacts under Title V. These demonstration projects simplified the contracting and compacting processes and made it easier for tribes to assume control of IHS programs. The self-governance demonstration projects mentioned above became permanent as Department of the Interior programs with PL 103-413 in 1994. In that same year, congressional legislation extended the concept of self-determination to include all IHS services on a trial basis. Tribes were allowed to contract with the IHS for funds to develop their own health services and assume control of programs that were formerly developed and controlled by the IHS and BIA (Berry et al. 2004; IHS 2005).

Tribes successfully managed the funds and programs contracted and preferred the empowerment of the arrangement. The successful outcomes of the trial projects resulted in legislation making the arrangement permanent. In 2000, tribal contracting and compacting of IHS programs became permanent with the addition of Title V (PL 106-260) to the Self-Determination Act. The IHCIA has been reauthorized four times since 1976 (Berry et al. 2004; IHS 2005, 2008). In 2004 the Potawatomi assumed control of their health clinic under the

compacting/contracting rules of the Indian Self-Determination Act. In 2010 the act was made permanent.

In 1995 the IHS Division of Behavioral Health (DBH) was established by the combination of two programs originally created independently in the 1970s. The Alcoholism and Substance Abuse Program, and the Mental Health and Social Services Program were merged to become the DBH. In the prevailing view at the time, alcoholism, substance abuse, and mental health and social services were related and thus required similar and interrelated services (IHS 2005).

### **Structural Violence Continues**

While it may be suggested that creation of the IHS and the self-determination acts attempted to rectify the structural violence perpetrated on Native Americans into the 20<sup>th</sup> century (McGuire 1990), some have suggested that the violence continued in earnest. Creation of the IHS and passage of the Indian Sanitation Facilities Act (1959) were really prerequisites to termination, not attempts to improve Indian health simply for the good of doing so. Self-determination has not been without problems. After years of being excluded from planning and management decisions, many tribes lacked the necessary resources and expertise to successfully administer their own health services. Combined with the continued underfunding of Indian health and bureaucratic barriers to the successful implementation of self-determination legislation, some tribal leaders began to see self-determination legislation as just another way for the federal government to rid itself once and for all of its obligations to Indian tribes (Adams 2000). Some viewed self-determination as being more destructive of the special status of Indian tribes than termination. Unlike termination, self-determination did not require that federal obligations to tribes be paid before the special status could be terminated (Deloria 1985).

The U.S. government has failed to live up to its trust responsibilities, to support the interests of the Indian tribes, because government interests are often competing. Indian tribes have had little success in compelling the government to live up to its responsibilities. Despite self-determination legislation, the government has not granted the Indian tribes full participation in matters regarding the trust relationship (Stull 1984). Official rhetoric and legislation in support of improved Indian health and welfare have not been matched by outcomes. Unfulfilled promises are evidenced by government failure to create the necessary resources for an effective health care system for Indians, and by the presence of sociocultural and organizational barriers that persist and restrict Indian access to quality health care (Berry et al. 2004).

The government's conflict of interest in the Indian trust relationship arises as a result of other bureaus in the Department of the Interior often have interests that are in direct conflict with the BIA. The Bureau of Land Management, Bureau of Reclamation, Minerals Management Service, Office of Surface Mining, U.S. Fish and Wildlife Service, and the U.S. Geologic Service often act in a manner contradictory to the government's responsibility to the tribes (Stull 1984). Though the Indian Self-Determination Act is an important piece of legislation, it failed to address the needed changes in the BIA. As a result of the policies and programs of the BIA and the manner in which they are administered, tribal dependency upon the BIA increased after the ISDEAA and still persists (Stull, Schultz, and Cadue 1986).

The BIA and IHS are often subjects of Indian criticism and suspicion. The BIA has been the object of denigration among many Indian tribes, often presented as the prime case in point of the inept nature of government bureaucracy (Stull 1984). At Senate Select Committee hearings on Indian policy in 1977, Indians testified that the BIA purposely avoided implementing tenets of the Indian Self-Determination Act (Prucha 1984a). More ominously, the IHS has been

accused of performing forced sterilizations on Indian women during the 1960s and 1970s (Lawrence 2000). These stories and allegations are a part of Indian perceptions of the BIA and IHS today, leading to distrust and a lack of involvement of many Indian people in these organizations.

Funding for IHS is insufficient by every pertinent measure in every facet of dispensing health care services (Berry et al. 2004). In 2002, tribal leaders estimated that the IHS budget, based on needs, would have to be 6.5 times greater than the amount actually allocated (Roubideaux 2002). Per capita health care funding among Indians on reservations is less than half of that provided to those who receive Medicaid or are in prison (Duran 2005). Expenditures on Native American health care are only 60 percent of that spent on the general American public under regular health insurance plans. Expenditures for all other federal health care programs are greater than that of the IHS, on a per capita basis, and IHS funding is less than that for other aspects of Department of Health and Human Services funding. Additionally, yearly funding increases for IHS have failed to consider population increases and the rates of inflation for medical costs (Berry et al. 2004). IHS funding is not an entitlement, legally. Funding is appropriated by Congress each year, and should funds be exhausted before the year is up, no other funds are available. With no additional funding, services may be rationed, and contracted health services with non-IHS providers may be suspended altogether (Cunningham 1996). Inadequate funding stresses the effectiveness of the Indian health system and plays a major role in the health disparities seen in Indian communities (Roubideaux 2002:1402). Most troublesome, inadequate funding has been cited as a problem of Indian health care for some time. The Merriam Report stated in 1928 that “The fundamental explanation of these low standards in

the medical work of the Indian Service is lack of adequate appropriations.” (Merriam et al. 1928)

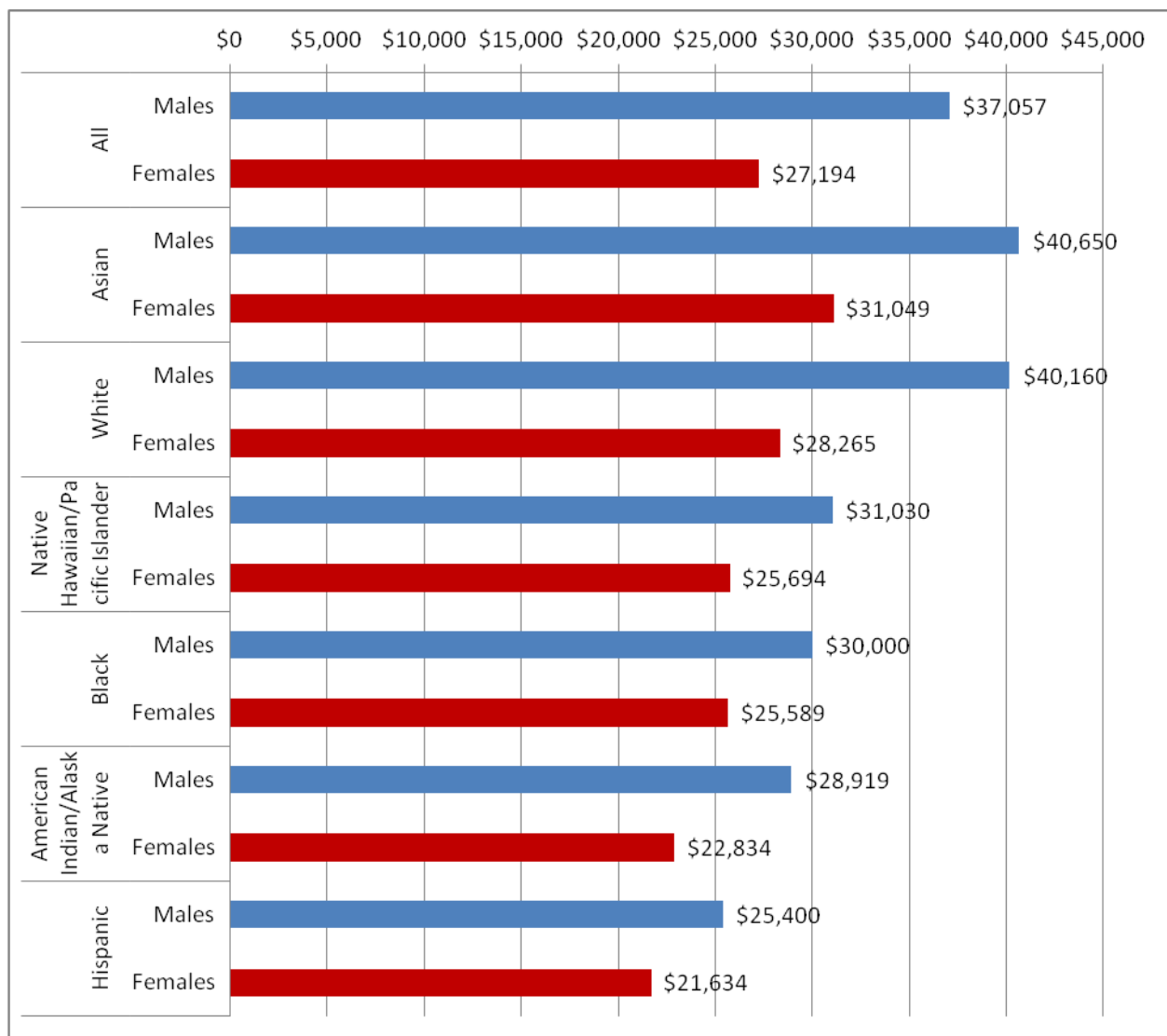
### *Poverty*

The reservation, assimilation, and termination periods impoverished Native Americans leaving them among the most economically disadvantaged racial groups in the U.S. Self-determination failed to provide significant and lasting economic benefits to many. The loss of land and resources and the lack of opportunities for economic development were major contributors to sustained poverty. Most Native Americans were poor throughout the 20<sup>th</sup> century and many remained poor even as they modernized (Hoxie 1996). The poor socioeconomic circumstances of Native Americans affect their living conditions, employment levels, the types of foods they are able to buy, exercise and recreational resources, and their overall physical and emotional well-being (Halpern 2007).

The 1988 Indian Gaming Regulatory Act legalized gaming as an economic resource for tribes. Indeed some Indian casinos have experienced success. Tribes with successful casinos have been able to improve housing, education, and health care; have revitalized cultural traditions; and have become more self-sufficient (Darian-Smith 2004). The National Indian Gaming Association (NIGC 2007) reported that net Indian gaming revenue was being spent as follows: 20% of net revenue was used for education, children and elders, culture, charity and other purposes; 19% went to economic development; 17% to health care; 17% to police and fire protection; 16% to infrastructure; and 11% to housing.

Many Indian casinos have not produced substantial revenue, however, and some have failed completely. Only around 30 percent of tribes have opened reservation casinos, and of those, only 15% generated over half of all total Indian gaming revenue. Success in gaming

seems to be partially dependent upon location. Tribes with casinos near large metropolitan areas fare better. Many reservations, and thus their casinos, are in isolated, rural areas that are removed from a regular clientele. The economic benefits of gaming are therefore experienced by a relatively small number of tribes and not equally distributed (Darian-Smith 2004). Thus, despite economic successes for some tribes through gaming, poverty continues to be an issue for much of the Native American population.



**FIGURE 4-1. Median income in the U.S. by race/ethnicity and sex, 1999. (U.S. Census Bureau, Census 2000 Summary File 3; Spraggins 2005).**



The median income of Native American males and females in 1999 still was the lowest of all racial groups except Hispanics (Figure 4-1). By the close of the 20<sup>th</sup> century, median income of Native Americans was only about 80% of that of the general U.S. population (Spraggins 2005).

Indian people today are approximately twice as likely as the general U.S. population to live in poverty, be unemployed, and lack a post-secondary education (Castor et al. 2006; Manson and Altschul 2004). Currently more than one-quarter of American Indian/Alaska Natives live in poverty. On average poverty among Native Americans was as high as 23% in 2001-2003 (versus 12.1% in the U.S. overall in 2002), with some tribes experiencing poverty rates as high as 40%

**TABLE 4-1. Labor force and unemployment statistics for Native Americans and U.S. whites, 1950 – 2000, by sex (Carter et al. 2006, Table Ag735-758; Fullerton 1999; U.S. Bureau of Labor Statistics, Labor Force Statistics from the Current Population Survey, 2010).**

Year	Both Sexes - Percentage In Labor Force		Males - Percentage In Labor Force		Females – Percentage In Labor Force	
	Native American	U.S. White	Native American	U.S. White	Native American	U.S. White
1950	41.1	59.2	63.0	86.4	17.0	33.9
1960	42.5	59.4	59.5	83.3	25.5	37.7
1970	49.0	60.4	63.4	79.7	35.3	43.3
1980	58.6	63.8	69.6	77.4	48.1	51.5
1990	62.1	66.5	69.4	76.4	55.1	57.5
2000	60.0	67.1	*	74.8	*	59.9
Year	Both Sexes - Civilian Labor Force - Percentage Unemployed		Males - Civilian Labor Force - Percentage Unemployed		Females - Civilian Labor Force - Percentage Unemployed	
	Native American	U.S. White	Native American	U.S. White	Native American	U.S. White
1950	12.1	5.3	13.0	5.1	8.8	5.7
1960	14.5	5.5	16.2	5.4	10.8	5.9
1970	11.1	4.9	11.6	4.4	10.2	5.9
1980	13.2	7.1	14.1	6.9	11.9	7.4
1990	14.4	5.6	15.4	5.7	13.1	5.5
2000	7.6	4.0	*	3.9	*	4.1

\*BLS does not provide data by sex for Native Americans.

(Sarche and Spicer 2008). Native Americans historically have had lower labor force participation and higher unemployment rates than the general U.S. population (Table 4-1): the rate of unemployment among Native Americans remains much higher than that of whites. Recent family unemployment rates have ranged from 14.4% to as high as 35% on some reservations (Sarche and Spicer 2008). Regarding discrepancies in education levels, significantly fewer native people (71%) earn a high school diploma or GED (80% nationally). Only 11.5% of Native Americans earning a bachelor's degree compared to 24.4% of the general U.S. population (Sarche and Spicer 2008).

Food and income insecurity are associated with reduced expenditures on food, lower consumption of fruits and vegetables, and less nutritious diets (Drewnowski and Specter 2004). Impoverished people more often suffer from poor nutrition and a whole spectrum of associated chronic conditions. Nutrition transition is related to other aspects of lifestyle such as cigarette smoking, disruption of social networks, reduction of physical activity, and greater stress. The major health trend among Native American groups has been toward obesity, diabetes, and diabetic complications such as cardiovascular disease. These conditions are more prevalent among Native Americans in North America than among any other racial subgroup. This situation is due to changing dietary patterns and reduced activity. Increased intake of saturated fat, simple sugars, and alcohol in modern diets has resulted in a greater incidence of gall bladder disease, tooth loss, and alcoholism and associated conditions such as fetal alcohol syndrome (Kuhnlein and Receveur 1996).

The rural location of many Indian reservations exacerbates food and income insecurity. Rural grocery stores are generally fewer, smaller, and offer less variety. Amenities for physical activity, such as fitness centers, walking trails, sidewalks, and sporting leagues are less

accessible. Clinics and hospitals are often smaller and dispersed. Additionally, the distances needed to travel for any services are a burden, particularly for those lacking reliable transportation (Casey et al. 2008; Smith and Morton 2009).

## **Summary and Conclusion**

Health disparities among Native Americans existed in the past and persist in to the present. The causes behind the disparities include governmental policies and their implementation. Treaties established the reservation system and provided the basis for federal responsibility for Indian health care—a responsibility that the government often views as an expensive burden. Assimilation policies such as allotment impoverished Indians. With few economic resources, tribes had only inadequately funded government health services on which to rely. Termination attempted to eliminate the special status of Indian people altogether, and many tribes suffered the loss of funding and services that resulted from termination. Despite self-determination legislation, health care services for Native Americans are still dependent upon federal appropriations. Structural violence has worked directly through the institutions and federal policies determining Indian affairs.

Indian policy has featured multifarious directives due to contradictory interests and competing philosophies in the federal government, as well as to shifts in the prevailing beliefs regarding governmental responsibilities to Native Americans. Opposing camps in government often supported similar policies, such as reservations and assimilation, albeit for different reasons. The result has been incongruous Indian policies that contribute to health disparities among Native American tribes.

Cultural and biological explanations of Indian health are often viewed as competing. It is most likely, however, that the specific pattern of epidemiologic transition and observed health

disparities of Native Americans result from a complex interaction of both factors. A more complete understanding of the health of Native Americans requires both biological and societal explanations. Such a biocultural approach may offer a more realistic explanation of Native American epidemiology.

## Chapter 4 References Cited

- Adams, A. 2000. The Road Not Taken: How Tribes Choose between Tribal and Indian Health Service Management of Health Care Resources. *American Indian Culture and Research Journal* 24(3):21-38.
- Adams, David W. 1995. *Education for Extinction: American Indians and the Boarding School Experience, 1875-1928*. Lawrence, Kansas: University Press of Kansas.
- Barker, D.J.P. 1994. *Mothers, Babies, and Disease in Later Life*. London: BMJ Publishing.
- . 1997. Maternal Nutrition, Fetal Nutrition, and Disease in Later Life. *Nutrition* 13(9):807-813.
- Barsh, Russel L. 1991. Progressive-Era Bureaucrats and the Unity of Twentieth-Century Indian Policy. *American Indian Quarterly* 15(1):1-17.
- Barsh, Russel L., and Diaz-Knauf, K. 1984 The Structure of Federal Aid for Indian Programs in the Decade of Prosperity, 1970 – 1980. *American Indian Quarterly* 8(1):1-35.
- Bee, Robert L. 1999. Structure, Ideology, and Tribal Governments. *Human Organization* 58:285-294.
- Benysheck, D.C. 2001. *The Political Ecology of Diabetes Among the Havasupai Indians of Northern Arizona*. PhD Dissertation. Arizona State University.
- Benysheck, D.C. and Watson, J.T. 2006. Exploring the Thrifty Genotype's Food-Shortage Assumptions: A Cross-Cultural Comparison of Ethnographic Accounts of Food Security among Foraging and Agricultural Societies. *American Journal of Physical Anthropology* 131(1):120-126.
- Bergman, A.B., Grossman, D.C., Erdrich, A.M., Todd, J.G., and Forquera, R. 1999. A Political History of the Indian Health Service. *The Milbank Quarterly* 77:571-604.
- Berry, Mary F., Reynoso, C., Braceras, J.C., Edley, Jr., Kirsanow, P.N., Meeks, E.M., Redenbaugh, R.G., and Thernstrom, A. 2004. *Broken Promises: Evaluating the Native American Health Care System*. U.S. Commission on Civil Rights. Washington, D.C.: U.S. Government Printing Office.
- Bird, A. 2007. Perceptions of Epigenetics. *Nature* 447(24): 396-398.
- Bloomgarden, Z.T. 1998. Insulin Resistance: Current Concepts. *Clinical Therapeutics* 20(2): 216-231.
- Brophy, William A., and Aberle, Sophie D. 1966. *The Indian, America's Unfinished Business: Report of the Commission on the Rights, Liberties, and Responsibilities of the American Indian*. Norman: University of Oklahoma Press.

Burt, Larry W. 1986. Roots of the Native American Urban Experience: Relocation Policy in the 1950s. *American Indian Quarterly* 10(2):85-99.

Cahn, Edgar S., ed. 1970. *Our Brother's Keeper: The Indian in White America*. Edited by Jerry Berman, W. Dayton Coles, Jr., Nancy Esposito, F. Browning Pipestem, assoc. New York: New Community Press.

Caldwell, Russell L. 1956. Is There an American Indian Policy? *Ethnohistory* 3(2):97-108.

Campbell, G.R. 1994. "Indian Health Service," in *Native America in the Twentieth Century: An Encyclopedia*. Mary Davis, ed. Pp. 256-261. New York: Garland Publishing.

Casey, A., Elliot, M., Glanz, K., Haire-Joshu, D., Lovergreen, S., Saelens, B., Sallis, J., and Brownson, R. 2008. Impact of Food Environment and Physical Activity Environment on behaviors and Weight Status in Rural U.S. Communities. *Preventive Medicine* 47:600-604.

Casper, M.L., Denny, C.H., Coolidge, J.N., Williams, G.I., Crowell, A., Galloway, J.M., and Cobb, N. 2005. *Atlas of Heart Disease and Stroke Among American Indians and Alaska Natives*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention and Indian Health Service.

Castor, Mei L., Smyser, Michael S., Taulii, Maile M., Park, Alice N., Lawson, Shelley A., and Forquera, Ralph A. 2006. A Nationwide Population-Based Study Identifying Health Disparities Between American Indians/Alaska Natives and the General Populations Living in Select Urban Counties. *American Journal of Public Health* 96:1478-1484.

Clifton, James A. 1998. *The Prairie People: Continuity and Change in Potawatomi Indian Culture, 1665 - 1965*. Iowa City: University of Iowa Press,

Crews, D.E. and Gerber, L. 1994. "Chronic Degenerative Diseases and Aging," in *Biological Anthropology and Aging*. Edited by D. Crews and R. Garruto, pp. 154-181. New York: Oxford University Press,

Dabelea, D., Hanson, R.L., Lindsay, R.S., Pettitt, D.J., Imperatore, G., Gabir, M.M., Roumain, J., Bennett, P.H., and Knowler, W.C. 2000. Intrauterine Exposure to Diabetes Conveys Risks for Type 2 Diabetes and Obesity: A Study of Discordant Sibships. *Diabetes* 49: 2008-2211.

Darian-Smith, E. 2004. *New Capitalists: Law, Politics, and Identity Surrounding Casino Gaming on Native American Land*. Belmont, Calif.:Wadsworth/Thomson.

Defronzo, R.A., and Ferrannini, E. 1991. Insulin Resistance: A Multifaceted Syndrome Responsible for NIDDM, Obesity, Hypertension, Dyslipidemia, and Atherosclerotic Cardiovascular Disease. *Diabetes Care* 14: 173-194.

Deloria, Vine 1985. "The Evolution of Indian Policy Making," in *American Indian Policy in the Twentieth Century*. Edited by Vine Deloria, pp. 239-256. Norman: University of Oklahoma Press.

Demerath, E.W., Cameron, N., Gillman, M.W., Towne, B., and Siervogel, R.M. 2004. Telomeres and Telomerase in the Fetal Origins of Cardiovascular Disease: A Review. *Human Biology* 76(1):127-146.

- Farmer, P.E. 2004. The Anthropology of Structural Violence. *Current Anthropology* 45(3):305-325.
- Farmer, P.E., Nizeye, B., Stulac, S., and Keshavjee, S. 2006. Structural Violence and Clinical Medicine. *PLoS Medicine* 3(10):1686-1691.
- Fiorentino, Daniele 1999. *Acculturation/Assimilation: American Indian Policy in the Progressive Years*. Ph.D. dissertation, Department of History, University of Kansas.
- Fox, S.I. 2011. *Human Physiology*, 12<sup>th</sup> Edition. New York: McGraw-Hill.
- Fritz, H.E. 1963. *The Movement for Indian Assimilation, 1860-1890*. Philadelphia: University of Pennsylvania Press.
- Garrett, J.T. 1994. "Health," in *Native America in the Twentieth Century: An Encyclopedia*. Edited by Mary Davis, pp. 233-237. New York: Garland Publishing.
- Getches, D. H., Wilkinson, C.F., and Williams, R.A. 2004. *Cases and Materials on Federal Indian Law*, 5th ed. American Casebook Series. St. Paul, Minn.: Thomson-West.
- Gerber, L.M. and Crews, D.E. 1999. "Evolutionary Perspectives on Chronic Degenerative Diseases," in *Evolutionary Medicine*. Edited by W. Trevathan, pp. 443-470. New York: Oxford University Press.
- Gilligan, J. 1996. *Violence: Reflections on a National Epidemic*. New York: Putnam.
- Gluckman, P.D., Hanson, M.A., Cooper, C., and Thornburg, K.L. 2008. Effect of In Utero and Early-Life Conditions on Adult Health and Disease. *The New England Journal of Medicine* 359(1): 61-73.
- Gluckman, P.D., Hanson, M.A., and Spencer, H.G. 2005. Predictive Adaptive Responses and Human Evolution. *Trends in Ecology and Evolution* 20(10): 527-533.
- Grinde Jr., Donald A. 2004. Taking the Indian out of the Indian: U.S. Policies of Ethnocide through Education. *Wicazo Sa Review* 19(2):25-32.
- Guthrie, M.C. 1929. The Health of the American Indian. *Public Health Reports* 44:945-957.
- Hales, C.N., and Barker, D.J. 1992. Type 2 (Non-Insulin-Dependent) Diabetes Mellitus: The Thrifty Phenotype Hypothesis. *Diabetologia* 35(7):595-601.
- . 2001. The Thrifty Phenotype Hypothesis. *British Medical Bulletin* 60:5-20.
- Halpern, P. 2007. *Obesity and American Indians/Alaska Natives*. Report Prepared for the U.S. Department of Health and Human Services, Office of the Assistant Secretary for Planning and Evaluation.
- Himsworth, H. 1936. Diabetes Mellitus: a Differentiation into Insulin-Sensitive and Insulin-Insensitive Types. *Lancet* 1:127-130.
- Hoxie, F.E. 1996. *Encyclopedia of North American Indians*. Boston: Houghton Mifflin.
- IHS (Indian Health Service). 2005. *The First 50 Years of the Indian Health Service: Caring and Curing*. Rockville, Md.: U.S. Department of Health and Human Services.

—. 2008. Indian Health Care Improvement Act. The Indian Health Service Fact Sheets. <http://info.ihs.gov/HlthImprvAct.asp> November 1st, 2008.

Joe, Jennie R. 1991. "The Delivery of Health Care to American Indians: History, Policies, and Prospects," in *American Indians: Social Justice and Public Policy*. Volume 9. Edited by Donald E. Green and Thomas V. Tonnesen, pp. 149-180. Ethnicity and Public Policy Series. Madison: University of Wisconsin.

Johnson, T.R., Champagne, D., and Nagel, J. 1997. "American Indian Activism and Transformation: Lessons from Alcatraz," in *American Indian Activism: Alcatraz to the Longest Walk*. Edited by T.R. Johnson, J. Nagel, and D. Champagne, pp. 9-44. Champaign, Illinois: University of Illinois Press.

Josephy, Alvin M., Nagel, J. and Johnson, Troy R. 1999. *Red Power: The American Indians' Fight for Freedom*. Lincoln: University of Nebraska Press.

Kanaka-Gantenbein, C. 2010. Fetal origins of adult diabetes. *Annals of the New York Academy of Science* 1205(1):99-105.

Kane, Robert L., and Kane, Rosalie A. 1972. *Federal Health Care (With Reservations!)*. New York: Springer Publishing Company.

Kelly, C.G. 1952. *Welfare Aspects of the Potawatomi Indian Agency*. Master's Thesis, The University of Kansas.

Kennedy, Edward M., Yarborough, R., Williams, H.A., Mondale, W.F., Hughes, H.E., Dominick, P.H., Murphy, G., Saxbe, W.B., Smith, R.T. and Parmeter, A.L. 1969. *Indian Education: A National Tragedy – A National Challenge*. Special Subcommittee on Indian Education Pursuant to S. Res. 80, A Resolution Authorizing an Investigation into the Problems of Education for American Indians. Washington, D.C.: U.S. Government Printing Office.

Knowler, W.C., Pettitt, D.J., Bennett, P.H., and Williams, R.C. 1983. Diabetes Miletus in the Pima Indians: Genetic and Evolutionary Considerations. *American Journal of Physical Anthropology* 62(1):107-114.

Kunitz, S.J. 2004. "The Evolution of Disease and the Devolution of Health Care for American Indians," in *The Changing Face of Disease: Implications for Society*. Edited by N. Mascie-Taylor, J. Peters, and S. McGarvey, pp. 153-169. Society for the Study of Human Biology Series: 43. Boca Raton, FL: Routledge.

Kuzawa, C.W. and Sweet, E. 2009. Epigenetics and the Embodiment of Race: Developmental Origins of US Racial Disparities in Cardiovascular Health. *American Journal of Human Biology* 21:2–15.

Kuzawa, C.W. and Thayer, Z.M. 2011. Timescales of Human Adaptation: The Role of Epigenetic Processes. *Epigenomics* 3(2):221-234.

Lappe, M. 1994. *Evolutionary Medicine: Rethinking the Origins of Disease*. San Francisco: Sierra Club Books.

- McCance, D.R., Pettitt, D.S., Hanson, R.L., Jacobsson, L.T., Knowler, W.C., and Bennett, P.H. 1993. Birthweight and Non-Insulin-Dependent Diabetes: Thrifty Genotype, Thrifty Phenotype, or Surviving Small Baby Genotype? *British Medical Journal* 308:942-945.
- McGarry, J.D. 2002. Dysregulation of Fatty Acid Metabolism in the Etiology of Type 2 Diabetes. *Diabetes* 51(1):7-18.
- McGuire, Thomas R. 1990. Federal Indian Policy: A Framework for Evaluation. *Human Organization* 49:206-216.
- Meriam, L., Brown, R.A., Roe Cloud, H., Dale, E.E., Duke, E., Edwards, H.R., McKenzie, F.A., Mark, M.L., Ryan Jr., W.C., and Spillman, W.J. 1928. *The Problem of Indian Administration*. Report of a Survey at the Request of Honorable Hubert Work, Secretary of the Interior, and Submitted to Him, February 21, 1928. Washington, D.C.: Institute for Government Research.
- NDIC (National Diabetes Information Clearinghouse). 2006. *Insulin Resistance and Pre-Diabetes*. National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health (NIH). Washington, DC: U.S. Department of Health and Human Services.
- Neel, J.V. 1962. Diabetes mellitus: A Thrifty Genotype Rendered Detrimental by Progress? *American Journal of Human Genetics* 14:353-62.
- . 1982. "The Thrifty Gene Revisited," in *The Genetics of Diabetes Mellitus*. Edited by J. Koberling and R. Tattersall, pp. 283-293. New York: Academic Press.
- O'Fallon, B., and Fehren-Schmitz, L. 2011. Native Americans Experienced a Strong Population Bottleneck Coincident with European Contact. *PNAS* published ahead of print December 5, 2011, doi:10.1073/pnas.1112563108. <http://www.pnas.org/content/early/2011/11/28/1112563108.full.pdf+html> December 27<sup>th</sup>, 2011.
- Oken, E. and Gillman, M.W. 2003. Fetal Origins of Obesity. *Obesity Research* 11:496-506.
- Perrott, George St. J. and West, M.D. 1957. Health Services for American Indians. *Public Health Reports (1896-1970)* 72(7): 565-570.
- Pratt, R.H. 1973. "The Advantages of Mingling Indians with Whites," in *Americanizing the American Indians: Writings by the "Friends of the Indian" 1880-1900*. Edited by F. Prucha, pp. 260-271. Cambridge, Mass.: Harvard University Press.
- Prentice, A.M., Hennig, B.J., and Fulford, A.J. 2008. Evolutionary Origins of the Obesity Epidemic: Natural Selection of Thrifty Genes or Genetic Drift Following Predation Release? *International Journal of Obesity* 32:1607-1610.
- Prucha, Francis P. 1976. *American Indian Policy in Crisis: Christian Reformers and the Indian, 1865 - 1900*. Norman, OK: University of Oklahoma Press.
- . 1984. American Indian Policy in the Twentieth Century. *The Western Historical Quarterly* 15(1):4-18.
- . 1997 *American Indian Treaties: The History of a Political Anomaly*. Berkeley: University of California Press.



- . 2000. *Documents of United States Indian Policy*, 3rd edition. Lincoln: University of Nebraska Press.
- Putney, Diane T. 1980. *Fighting the Scourge: American Indian Morbidity and Federal Policy, 1897 – 1928*. PhD Dissertation. Milwaukee, WI: Marquette University.
- Reinhard, K.J., Johnson, K.L., LeRoy-Toren, S., Wieseman, K., Teixeira-Santos, I., and Vieira, M. 2012. Understanding the Pathoecological Relationship between Ancient Diet and Modern Diabetes through Coprolite Analysis. *Current Anthropology* 53(4):506-512.
- Rhoades, Everett R., Luana L. Reyes, L.L., and Buzzard, G.D. 1987. The Organization of Health Services for Indian People. *Public Health Reports* 102:352-356.
- Ritenbaugh, C. and Goodby, C.S. 1989. Beyond the Thrifty Gene: Metabolic Implications of Prehistoric Migration into the New World. *Medical Anthropology* 11(3):227-236.
- Rosenbloom, A.L., Joe, J.R., Young, R.S., and Winter, W.E. 1999. Emerging Epidemic of Type 2 Diabetes in youth. *Diabetes Care* 22(2):345-354.
- Shaffer, E.A. 2005. Epidemiology and Risk Factors for Gallstone Disease: Has the Paradigm Changed in the 21st Century? *Current Gastroenterology Reports* 7(2):132-140.
- Shen, S., Reaven, G.M., and Farguhar, J.W. 1970. Comparison of Impedance to Insulin-Mediated Glucose Uptake in Normal Subjects with Latent Diabetes. *The Journal of Clinical Investigation* 49(12):2151-2160.
- Smith, C. and Morton, L.W. 2008. Rural Food Deserts: Low-Income Perspectives on Food Access in Minnesota and Iowa. *Journal of Nutrition Education and Behavior* 41(3):176-187.
- Smith-Morris, Carolyn M. 2004 Reducing Diabetes in Indian Country: Lessons from the Three Domains Influencing Pima Diabetes. *Human Organization* 63:34-46.
- Speakman, J.R. 2006. Thrifty Genes for Obesity and the Metabolic Syndrome – Time to Call off the Search? *Diabetes and Vascular Disease Research* 3(1):7-11.
- . 2008. Thrifty Genes for Obesity, an Attractive but Flawed Idea, and an Alternative Perspective: the ‘Drifty Gene’ Hypothesis. *International Journal of Obesity* 32:1611-1617.
- Stöger, R. 2008. The Thrifty Epigenotype: An Acquired and Heritable Predisposition for Obesity and Diabetes? *BioEssays* 30(2):156-166.
- Stremlau, R. 2005 To Domesticate and Civilize Wild Indians: Allotment and the Campaign to Reform Indian Families, 1875-1887. *Journal of Family History* 30:265-286.
- Stuart, Paul H. 1990 Financing Self-Determination: Federal Indian Expenditures, 1975-1988. *American Indian Culture and Research Journal* 14:1-18.
- Stull, Donald D. 1984. *Kiikaapoa: The Kansas Kickapoo*. Horton, KS: Kickapoo Tribal Press.
- . 1990. Reservation Economic Development in the Era of Self-Determination. *American Anthropologist* 92:206-210.

- Stull, Donald D., Schultz, Jerry A., and Cadue, Ken Sr. 1986. Rights without Resources: The Rise and Fall of the Kansas Kickapoo. *American Indian Culture and Research Journal* 10(2):41-59.
- Szathmary, E.J. 1990. "Diabetes in Amerindian Populations: The Dogrib Studies," in *Disease in Populations in Transition: Anthropological and Epidemiological Perspectives*. Edited by A. Swedlund and G. Armelagos, pp. 75-103. New York: Bergin and Garvey.
- Ten, S. and Maclaren, N. 2004. Insulin Resistance Syndrome in Children. *Journal of Clinical Endocrinology & Metabolism* 89(6):2526-2539.
- Tyroler, H.A. and Patrick, R. 1972 Epidemiologic Studies of Papago Indian Mortality. *Human Organization* 31:163-170.
- Tyler, S. Lyman 1973. *A History of Indian Policy*. Washington, D.C.: Bureau of Indian Affairs, United States Department of the Interior, U.S. Government Printing Office.
- U.S. Congress. 1986. *Indian Health Care*. OTA-H-290, Office of Technology Assessment. Washington, D.C.: U.S. Government Printing Office.
- Walch, Michael C. 1983. Terminating the Indian Termination Policy. *Stanford Law Review* 35:1181-1215.
- Wareharn, N.J. 2004. "Unraveling Gene-Environment Interactions in Type 2 Diabetes," in *The Changing Face of Disease: Implications for Society*. Edited by N. Mascie-Taylor, J. Peters, and S. McGarvey, pp. 130-138. Society for the Study of Human Biology Series: 43. Boca Raton, FL: Routledge.
- Washburn, Wilcomb, E. 1984. A Fifty Year Perspective on the Indian Reorganization Act. *American Anthropologist* 86:279-289.
- Weiss, K.M. 1990. "Transitional Diabetes and Gallstones in Amerindian Peoples: Genes or Environment?" in *Disease in Populations in Transition: Anthropological and Epidemiological Perspectives*. Edited by A. Swedlund and G. Armelagos, pp. 105-123. New York: Bergin and Garvey.
- Weiss, K.M., Ferrell, R.E., and Hanis, C.L. 1984. A New World Syndrome of Metabolic Diseases with a Genetic and Evolutionary Basis. *Yearbook of Physical Anthropology* 27:153-178.
- Weiss, K.M., Ulbrecht, J.S., Cavanagh, P.R., and Buchanan, A.V. 1989. Diabetes Mellitus in American Indians: Characteristics, Origins, and Preventive Health Care Implications. *Medical Anthropology* 11(3):283-304.
- Wendorf, M. 1989. Diabetes, the Ice Free Corridor, and the Paleoindian Settlement of North America. *American Journal of Physical Anthropology* 79:503-520.
- Wendorf, M. and Goldfine, I.D. 1991. Archaeology of NIDDM. Excavation of the Thrifty Genotype. *Diabetes* 40(2):161-165.
- Whitney, E. and Rolfes, S.R. 2011. *Understanding Nutrition*, 12<sup>th</sup> Edition. Belmont, CA: Wadsworth Cengage Learning.

Wilkins, D. E. 1997. *American Indian Sovereignty and the U.S. Supreme Court: The Masking of Justice*. Austin: University of Texas Press.

Williams, G.C. 1957. Pleiotropy, Natural Selection, and the Evolution of Senescence. *Evolution* 11:398–411.

Yilmaz, N., Kilic, S., Kanat-Pektas, M., Guleman, C., and Mollamahmutoglu, L. 2009. The Relationship between Obesity and Fecundity. *Journal of Women's Health* 18(5):633-636.

Young, T.K. 1994. *The Health of Native Americans: Toward a Biocultural Epidemiology*. Oxford: Oxford University Press.

## **Chapter 5: The Epidemiologic Transition in Native American Populations in the 20<sup>th</sup> Century**

Omran (1971) did not reference Native Americans in his original papers on the epidemiologic transition. Nonetheless there have been substantial improvements in the last 50 years in Native American infant mortality, life expectancy and incidence of infectious diseases, particularly tuberculosis. Native Americans have experienced the epidemiologic transition and entered Omran's *Age of Degenerative and Man-Made Diseases*, in which infectious diseases decline and chronic diseases predominate (Young 1994, 1996). The pattern of Native American disease experience through time has been referred to as an "unnatural history" (Campbell 1989), featuring the preeminence of infectious diseases, followed by a dramatic rise of degenerative diseases, and the development of social pathologies in recent decades (Young 1988).

### **Native American Health Data**

Throughout much of the 20th century, health data were only haphazardly collected for Native Americans. The development of the U.S. Census Bureau during the 20th century was a product of the growing governmental statistical system. Data for American Indians also became more precise over the century. A large amount of data for American Indians was finally available at the end of the 20<sup>th</sup> century, with much less at the beginning of the century and virtually none in the decades in between. The causes of this midcentury void and later recovery are complex. Popular opinions, official public policy, and politics had a significant impact on how American Indians were included in federal data collection and dissemination efforts (Carter and Sutch 2006).

The Meriam Report of 1928 cited the lack of accurate data on Indian health as a significant failure on the part of the BIA. At the time, the lack of data made it virtually

impossible to properly plan or execute effective health programs. Complaints regarding the lack of data continued into the 1930s and confirmed that no accurate data were collected or filed on the incidence or prevalence of tuberculosis or other diseases among Native Americans (McMillen 2008; Meriam et al. 1928).

Prior to 1955, most health statistics on Native Americans were the result of specific U.S. Senate or House committee initiatives designed to answer a particular question or to address a perceived problem. Although they did not constitute regular and systematic data collection, the results of the initiatives often called for such data collection to be instated (see the Meriam Report). Not until the IHS was established as a part of the Public Health Service would data be collected systematically and at prescribed intervals (Carter and Sutch 2006). Only in 1966 did the IHS begin to develop a comprehensive data information system. Called Operation SAM (Systems Analysis Module), San Xavier Indian Health Center at the Papago Service Unit near Tucson received the assignment to develop the first such system (Hackenberg 1966). Regardless, even as late as 1977, the Indian Policy Review Commission of the U.S. Congress complained about the lack of reliable data, citing the difficulty of obtaining data scattered across various federal agencies. Even when the data were obtainable, the commission found them to be incomplete and unrepresentative (Abourezk et al. 1977).

Census Bureau data do provide deaths by cause for Native Americans for select years, as do data from the National Office of Vital Statistics (NOVS) and National Center for Health Statistics (NCHS). Mortality statistics prior to 1921 are generally not available for Native Americans specifically. Prior to the establishment of the IHS, morbidity and mortality information was recorded by BIA area agencies in an unsystematic and inconsistent manner, if at all.

## **Trends in Native American Health**

### **Late 19<sup>th</sup> and Early 20<sup>th</sup> Centuries**

Much of the health information on Native Americans from the late 19<sup>th</sup> and early part of the 20<sup>th</sup> centuries consists of anecdotal descriptions in letters or reports from agency superintendents and other employees. Conditions on reservations exacerbated outbreaks of infectious diseases. After 1880, diseases related to poverty became prevalent and remained that way among many groups until 1940. Among Plains Indian groups, malnutrition became widespread after the bison were exterminated. Forced onto reservations, these groups were dependent upon the government to provide food. The provisioning was often inadequate and governmental promises of supplies, made by treaty, were often not kept (Lux 2001). Settlement into permanent housing facilitated the spread of pathogens that killed at high rates. Reservation homes were cramped wooden structures very different from traditional homes. The new housing placed people together at close quarters, with little knowledge of sanitary and hygienic practices that could prevent disease outbreaks (Trafzer 1997). During this time, concerns regarding Indian health focused on outbreaks of communicable diseases of poverty including influenza, tuberculosis, smallpox, whooping cough, measles, and trachoma (Carter 1916; Connelley 1918; USPHS 1913).

Tuberculosis began to pose a significant threat to Indian morbidity and mortality in the latter half of the 19<sup>th</sup> century and continued to do so into the early part of the 20<sup>th</sup> century. Confinement on reservations and unsanitary, crowded living conditions contributed to the high rate of tubercular infections. The Office of Indian Affairs also recognized trachoma as a significant health problem by 1908. Though not a killer like tuberculosis and smallpox, it was a source of severe debilitation. Trachoma caused irritation and a great deal of eye pain, hence the

common name, "sore eyes." The disease is caused by the microorganism *Chlamydia trachomatis* and is communicable through eye-to-eye or eye-to-hand contact, use of infected bedding, towels, handkerchiefs, and flies (Keller 2002; Putney 1980). Trachoma is characterized by the appearance of coarse granules on the inner surface of the eyelids, causing extreme irritation and scarring that can eventually lead to blindness.

### **1920s and 1930s**

Health conditions on Native American reservations became a greater federal concern in the 1920s, resulting in the Meriam Report in 1928. As federal officials became increasingly aware of the great health disparity between Native Americans and the rest of the country, more data on health conditions among Indians became available as data collection efforts increased. Cholera, dysentery, typhoid fever, typhus, and tuberculosis are usually associated with crowded living conditions, poor sanitation, and urban poverty (Keller 2002). BIA policies were indicted by the Meriam Report as fostering a favorable environment for these diseases, while doing nothing substantive to defeat them (Meriam et al 1928). Trachoma also continued to be among the most common afflictions among Native Americans during this time (Putney 1980).

It was government policy to utilize Indian boarding schools to promote good hygiene and health practices among students. Advocates of this policy believed that newly enculturated students would take these practices back with them to the reservations and teach their families what they had learned. In fact, research suggests that while the campaigns such as those of the National Tuberculosis Association may have succeeded in Indian schools, they failed on the reservations due to the lack of direct action (Putney 1980).

The adoption of Euro-American agricultural methods was supposed to provide Native Americans with better nutritional intake, and some research suggests that nutrition may have

played a key role in the reduction of the incidence of tuberculosis on Indian reservations during the early 20<sup>th</sup> century (Putney 1980), although this is speculative. Dietary intake on reservations is poorly documented for this period. Some Native American populations received food rations of beef, sugar, and flour from the Office of Indian Affairs, but these rations were cut during the first decade of the 20th century (Clifton 1998). Indian Commissioner Sells' "Save the Babies" campaign during the second decade of the 20th century authorized additional rations, including sweetened condensed milk for Native American mothers to feed their children during weaning. These additional calories may have contributed to a drop in infant mortality at the time. More and better food has also been credited with causing the eventual reduction of tuberculosis on Indian reservations (Putney 1980). However, whereas the rations may have provided more energy, they did not provide a nutritionally balanced diet, but did change Indian dietary habits. Policies such as this, with unintended long-term negative consequences, are a principal characteristic of policies toward Native Americans throughout the century.

Mortality among Native Americans continued to be high into the 1930s. In 1935 there were a reported 119,223 Indians in Arizona, California, Colorado, Kansas, Mississippi, Nevada, New Mexico, North Carolina, Oklahoma, and Utah. Among this group the death rate from tuberculosis was 204.7 per 100,000 population. Next to tuberculosis, infant deaths were cited as among the most devastating to Indian lives in the 1930s, with 97 deaths per 100,000 among Indians under one year of age (Townsend 1938), versus 56.9 per 100,000 among non-Indians. However, neonatal deaths among Indians in 1930-1931 were only slightly higher than those among a non-Indian comparison population, 10.7% versus 9.01% (Sterling 1933). From 1930 to 1939 the mortality rate from whooping cough (pertussis) alone among Indians was more than six times greater than for whites (Dauer 1943). The disease is a contagious bacterial infection

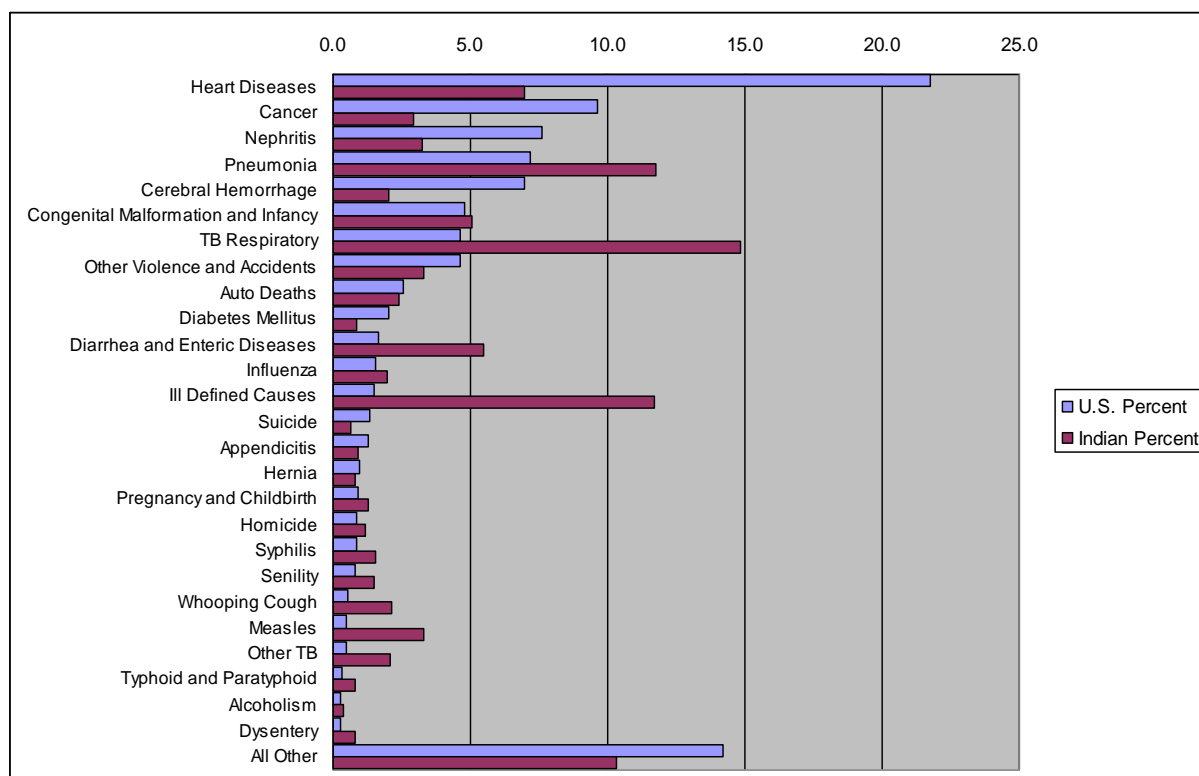


primarily of early childhood for which a vaccine was first developed in the 1920s (Baker and Katz 2004). Persistence of the condition in Native American populations is attributed to a lack of services in rural areas, lack of education on the condition, and insufficient distribution of the vaccine (Daur 1943).

Whereas infectious conditions affected Native Americans at high rates in the 1920s, chronic conditions affected them less frequently. Cancer rates were lower among Indians than whites during this period, with an average cancer mortality rate of 55 per 100,000 population, compared to 92.6 per 100,000 for all races in 1921-1925. By 1933 the rates were 42 per 100,000 for Indians versus 97.6 per 100,000 among whites. Two possible explanations for lower cancer rates among Indians in the 1920s-1930s have been suggested: either life expectancy was not as long as that of whites and therefore many Native Americans did not reach the advanced age when cancer is most prevalent; or many cases simply went undiagnosed (Townsend 1938). It is also possible that the traditional foods and the environment of the Native Americans on reservations did not expose them to as many carcinogens.

In a review of the trend for diabetes in Saskatchewan from 1905 to 1934, Chase (1937) concluded that Indians were not susceptible to diabetes. “Indians are not subject to diabetes. Complete physical examinations, including urinalyses, have been done on 1,500 Indians in Saskatchewan by the Anti-Tuberculosis League, and no case of diabetes has been discovered. Dr. A. B. Simes, who has charge of the medical work in the File Hills Reserve, has not seen a case of diabetes among Indians” (Chase 1937:369).

Causes of death by percent for the U.S. total population and Native Americans in 1934 are shown in Figure 5-1. Note the higher rates of heart disease and cancer deaths for the general



**FIGURE 5-1. Causes of death by percent for the U.S. total population and Native Americans, 1934. Ill-defined causes result from a lack of accurate diagnosis. Data adapted from Townsend (1938).**

U.S. population, but higher rates of pneumonia, diarrhea/enteric diseases, and respiratory tuberculosis for the Native Americans. The high percentage of Native Americans dying from “ill-defined causes” reflected the lack of trained medical personnel attending to the sick and dying on reservations and thus the lack of anyone to provide an accurate diagnosis (Townsend 1938). Native Americans also experienced a high number of deaths at younger ages compared to other populations in the U.S. (Table 5-1), with a particularly high percentage of deaths in the 1-19 age group. This pattern reflects the epidemiologic profile of Native Americans (shown in Figure 5-1) with high numbers of deaths from infectious diseases impacting the young.

Despite the continued assault on traditional institutions, efforts to improve Indian health began to show some success by the end of the fourth decade of the 20th century. By 1939 a

**TABLE 5-1. Percentage of total deaths (crude deaths) by age and race/ethnicity\*, United States: 1934 (Collinson 1936).**

<b>Age Group</b>	<b>All</b>	<b>White</b>	<b>Negro</b>	<b>Indian</b>	<b>Chinese</b>	<b>Japanese</b>	<b>Other</b>
< 1 year	9.33	8.52	12.88	21.62	5.57	9.02	29.71
1-19	7.68	7.04	10.49	24.39	6.81	17.83	19.65
20-44	16.48	14.25	30.07	19.33	24.15	21.97	24.06
45-64	27.41	27.39	28.65	13.85	31.06	43.84	15.86
65 and over	39.14	42.78	17.88	20.78	32.38	7.32	11.19

\*Race/ethnicity terms used by Collinson.

campaign to eradicate trachoma had been highly successful, a result of the discovery of sulfonamide as an effective treatment and the aggressive institution of sulfonamide therapy in Indian country. The Meriam Report, the end of allotment, and the Indian New Deal also had prompted the government to increase the number of Indian hospitals and medical staff (Castillo 1978; Tyler 1973).

### **1940s and 1950s**

Efforts to improve Indian health had begun to show some success by the beginning of the 1940s. By 1943 trachoma was no longer considered a major problem (Tyler 1973). Nonetheless other illnesses were still a scourge among Indians. John Collier retired from the BIA in 1945, and the New Deal policies of the 1930s gave way to new policies designed to end the special relationship between Indians and the federal government. For the first half of the 1940s the nation's attention was focused principally on World War II. Consequently, little effort was made to document health conditions among Native Americans, and very few data on morbidity and mortality were collected (Carter and Sutch 2006; Deloria 1985; Prucha 1984b; Tyler 1973).

The life expectancy of Native Americans in 1940 was approximately 52 years, much lower than that of other racial and ethnic groups in the United States. Indian infant mortality that same year was twice that of other racial and ethnic groups (Manson and Altschul 2004). Though

efforts to improve Indian health had begun to show success, some illnesses were still disproportionately affecting Indians. Between 10% and 25% of Indians drafted into the military during World War II were rejected for service because they had active tuberculosis (Bergman et al. 1999).

Following the war, the Hoover Commission was established to evaluate the administration of the federal government in general and to recommend changes. As a result of Hoover Commission recommendations, BIA health services were eliminated and regular tracking of Indian health conditions effectively ceased. Assimilation through the new termination policy was seen as the means by which Indian health would be improved and federal responsibility of Indian affairs could be brought to an end (Castillo 1978; Deloria 1985; Tyler 1973).

At mid-century half of all Indians were under the age of 20, compared with approximately 16.5% of the rest of the U.S. population. In 1953 most of the excess deaths of Indians were among the infants and children. Indian children under the age of five had a death rate more than twice that of the rest of the nation. Mortality rates for tuberculosis, pneumonia, influenza, and gastroenteritis were still much higher (Perrott and West 1957). Morbidity among Native Americans in 1953 still was characterized by high prevalence of eye and ear problems, dental disease, diarrhea, tuberculosis, and acute respiratory disease (Perrott and West 1957). Federal policy makers felt that the termination policy could not be achieved while Indian health status was so poor compared to the rest of the country.

As a means of addressing the health disparity so that complete termination could commence, the Transfer Act shifted Indian health care responsibilities to the Public Health Service (Kunitz 2004). At the time the Public Health Service assumed responsibility for Indian

health, living conditions on Indian reservations were described as promoting disease. The typical Indian family lived in crowded, substandard housing, many without safe drinking water or adequate waste disposal. Flies, rodents, and other disease vectors were a serious threat to health (Perrott and West 1957). As the 20th century progressed, government health officials had to acknowledge that high rates of tuberculosis among the Indians were an effect of rampant poverty rather than a “weak constitution,” as previously promoted by medical researchers and the health establishment (McMillen 2008).

A vast majority of Indians living on reservations were poor and had few opportunities to improve their economic standing, but wealthier Indians did not appear to have healthier habits. Education, time away from the reservation, and assimilation determined health and living conditions as much as wealth. Poverty among Indians in the 1950s resulted in a large proportion of them forgoing medical care in favor of essentials, but this behavior was not restricted to those Indians who were economically disadvantaged. Some with adequate incomes chose to make alternative purchases. Many Indians regarded free medical care as a service to which they were entitled by treaty regardless of economic status and thus chose not to seek any care for which they would have to pay (Perrott and West 1957).

**TABLE 5-2. Death rates per 1,000 population for selected races, by sex, 1940, 1950, 1960 (NCHS 1963).**

Race*	1940			1950			1960		
	Total	Male	Female	Total	Male	Female	Total	Male	Female
White	10.4	11.6	9.2	9.5	10.9	8	9.5	11	8
Negro	13.9	15.2	12.6	11.3	12.6	10	10.4	11.8	9
Indian	14.3	14.7	13.9	13	14	11.8	8.6	10.1	7.2
Chinese	15.3	18.3	6.7	9	11.6	4.2	6.8	9.8	2.9
Japanese	6.7	8.8	4.1	6.1	8.6	3.2	5.1	7	3.3
Other	9.4	8.7	13.3	3.6	4.6	1.7	5.4	6.7	3.6

\*Terms used by NCHS.

Public health officials recognized that when Native Americans adopted Euro-American habits, their epidemiological profile became more similar to that of the rest of the country. For instance, when Indian groups in South Dakota and Arizona became more acculturated, their patterns of age and sex at death became more like those of non-Indians (Wissler 1936). Table 5-2 shows that Native American death rates became more similar to those of whites in the period from 1940 to 1960, as tribes acculturated, and actually were lower by 1960.

### **1960s and 1970s**

In a study among Southwestern Indians from 1957 to 1964, Sievers (1966) found a high incidence of diabetes, particularly among those who tended toward obesity. Gastric and bile duct cancers were the primary types of cancer among Indians in the Southwest, whereas lung and breast cancers were less frequent than among whites. Atherosclerosis was rare, probably due to low cholesterol levels. Heart attacks seldom occurred among Indians without preexisting diabetes, high blood pressure, or other recognized risk factors. Hypertension was less common than among whites. Tuberculosis, however, persisted at a high rate among the Indians of the southwest at this time. Coccidioidomycosis (valley fever) was a problem among Indians who lived in or visited the area (western U.S and northern Mexico) where the fungus was present in the soil. Trachoma still was a common eye disease among these Indians, whereas open-angle (chronic) glaucoma was absent. Rheumatoid arthritis was prevalent, but asthma was not. Alcoholism, Laennec's cirrhosis, dietary deficiencies, and esophageal varices (dilated veins associated with portal hypertension due to cirrhosis) were major health problems for most of the area tribes. A high rate of trauma among the southwestern Indians was related to alcoholism, automobile and horse-riding accidents. This pattern was indicative of a population with a mixed epidemiologic profile, featuring both chronic and infectious conditions.

The disparity in infant mortality rates (IMR) between Native American and other U.S. races shrunk dramatically between 1955 and 1967 (Table 5-3). The disparity in IMR decreased from 34.8 per 1,000 births to 11.7 per 1,000 in this 11-year period, resulting in an infant mortality rate in 1965 that was nearly 60% lower than that in 1955 (Wallace 1972).

Neonatal and postneonatal mortality are often examined separately due to most neonatal mortality resulting from congenital conditions and delivery (endogenous causes), whereas post-neonatal mortality is more likely to be due to conditions or events that occur after delivery and more likely reflect environmental conditions, known as exogenous causes (Rowley and MacDorman 1994). Differences between Native Americans and the total U.S. population in neonatal death rates were minimal in 1960 and 1966 and, in some cases, Native Americans displayed lower rates (Table 5-4). Infant mortality rates, however, still were much higher among Native Americans, but the disparity decreased from 1961 to 1965. In 1960, the largest disparities were for respiratory diseases (difference of 7.5/1,000 births) and infectious and parasitic

**TABLE 5-3. Infant mortality rates (per 1,000 live births), 1955-1965 (Wallace 1972)**

<b>Year</b>	<b>Indian</b>	<b>U.S. All Races</b>	<b>Difference</b>
1955	61.2	26.4	34.8
1956	56.1	26.0	30.1
1957	58.2	26.3	31.9
1958	56.7	27.1	29.6
1959	46.7	26.4	20.3
1960	47.6	26.0	21.6
1961	42.3	25.3	17.0
1962	41.8	25.3	16.5
1963	42.9	25.2	17.7
1964	35.9	24.8	11.1
1965	36.4	24.7	11.7

**TABLE 5-4. Cause-specific neonatal and post-neonatal mortality rates per 1,000 aged <1 year among Native Americans compared to the general U.S. population, 1960-1966 (Hill and Spector 1971).\***

		Indian**	U.S. Total	Indian**	U.S. Total
		1960		1966	
Neonatal	Immaturity	4.5	4.5	3.1	3.6
	Ill-defined	2.0	1.7	3.1	2.7
	Postnatal asphyxia and atelectasis	3.0	4.5	2.5	3.8
	Congenital malformations	2.3	2.3	1.7	2.3
	Birth injuries	2.4	2.4	1.6	2.0
Post-neonatal	Respiratory diseases	10.3	2.8	7.1	2.5
	Digestive diseases	5.2	0.8	3.6	0.5
	Accidents	1.9	0.8	1.9	0.8
	Infectious and parasitic diseases	2.2	0.3	1.6	0.3
	Congenital malformations	1.6	1.3	1.3	1.1

\*Minor causes omitted.

\*\*Indian rates are for the 3-year periods 1965-67 and 1959-61; all other rates are for the single years 1966 and 1960.

diseases, which were over seven times more common among Native Americans, followed by digestive disease (six-and-a-half times more common). By 1966 the disparity in respiratory diseases had dropped to 4.6/1,000 births and infectious and parasitic diseases were reduced to just over five times more common; digestive diseases, however, had increased to over seven times the rate in the general population.

Infant mortality continued to decline from 1966 to 1971 for Native Americans and the U.S. as a whole (Table 5-5). Pneumonia, a disease most likely to kill during the postneonatal period, was the most common cause of infant deaths among Native Americans in 1969, whereas congenital anomalies, primarily a neonatal cause of death, were the principal cause of death that year for all U.S. infants (Table 5-6). By 1971, postneonatal deaths among Native Americans were lower than neonatal deaths, but Native American postneonatal deaths were still more than double than that of the overall U.S.



**TABLE 5-5. Infant mortality rates (per 1,000 live births), 1966-1971 (IHS 1974).**

Year	Indian		U.S. Total	
	Neonatal	Postneonatal	Neonatal	Postneonatal
1966	17.3	21.7	23.7	6.5
1967	15.3	16.9	22.4	5.9
1968	14.4	16.5	21.8	5.7
1969	-	-	20.7	5.4
1970	-	-	19.8	5.0
1971	12.3	11.2	19.2	4.9

**TABLE 5-6. Causes of infant deaths by percent, 1969 (IHS 1974).**

Cause	Indian*	U.S. Total
Pneumonia	14.9	9.3
Congenital Anomalies	9.7	15.1
Symptoms and Ill Defined	9.6	4.3
Asphyxia of Newborn	7.0	12.8
Immaturity, unqualified	7.0	11.9
Respiratory Distress Syndrome**	4.5	5.5
Hyaline Membrane Disease**	3.5	6.4
Complications of Pregnancy	3.7	7.1
Diarrheal Diseases	6.8	1.1
Accidents	4.5	3.2
Meningitis	2.7	0.7
Septicemia	2.2	1.0
Other	23.9	21.6
Total	100.0	100.0

\*Based on 3 year average 1968-1970.

\*\*These are essentially the same condition, with hyaline membrane disease being an older term.

Deaths due to tuberculosis declined dramatically in the years following the establishment of the IHS in 1955. Use of the BCG vaccine became widespread after World War II (Comstock 1994). In 1955 the death rate from TB among Native Americans was over five times that of the U.S. as a whole (Table 5-7). By 1971, the rate had been reduced by 84%. Despite this progress, the rate of TB among Native Americans continued to be many times higher than the national average.

**TABLE 5-7. Deaths due to tuberculosis, 1955-1971 (IHS 1974).**

<b>Year</b>	<b>Indian Deaths</b>	<b>Indian Death Rate</b>	<b>U.S. Rate</b>
1955	208	47.3	9.1
1956	171	40.2	8.4
1957	143	34.2	7.8
1958	138	31.5	7.1
1959	140	27.9	6.5
1960	98	25.1	6.1
1961	105	24.5	5.4
1962	137	25.3	5.1
1963	114	24.8	4.9
1964	103	21.6	4.3
1965	96	19.3	4.1
1966	85	15.4	3.9
1967	82	13.4	3.5
1968	71	12.8	3.1
1969	82	13.0	2.8
1970	-	-	2.7
1971	51	7.6	2.1

Native American maternal mortality rates plunged during the years 1958-1967 (Table 5-8). The pronounced fluctuations from year to year and low rate in 1967 may reflect the low overall number of Native American maternal deaths, which averaged only 15 per year, and had a high standard deviation. Regardless, the trend is primarily downward. The difference between the Indian and U.S. rates decreased from 45 per 100,000 in 1958 to 5.9 per 100,000 in 1967.

Except for heart disease, cancers, strokes, and some diseases of early infancy, all of which were increasing, the age-adjusted death rates among Indians still were significantly higher than those of the general population. In 1971 the Indian postneonatal death rate was 2.3 times greater than that of the U.S. rate across all races (from Table 5-5). Additionally the tuberculosis death rate was 4.6 times greater, the influenza and pneumonia rate was 1.7 times greater, the rate for accidents was 3.3 times greater, and for cirrhosis, the rate was 4.7 times greater (Guyon

**TABLE 5-8. Comparison of Maternal mortality rates per 100,000 live births among American Indians and the general U.S. Population, 1958-1967 (Hill and Spector 1971).**

Year	American Indian	U.S. Total
1958	82.6	37.6
1959	68.8	37.4
1960	67.9	37.1
1961	66.5	36.9
1962	89.7	35.2
1963	83.7	35.8
1964	74.2	33.3
1965	63.4	31.6
1966	54.6	29.1
1967	33.9	28.0
<i>sd</i>	<i>15.3</i>	<i>3.4</i>

**TABLE 5-9. Comparison of life expectancy at birth, at 1 year and at age 5 for American Indians and the general U.S. population (Hill 1970).**

YEAR	AMERICAN INDIAN			U.S. TOTAL POPULATION		
	At Birth ( <i>e</i> <sub>0</sub> )	At 1 Yr ( <i>e</i> <sub>1</sub> )	At 5 Yrs ( <i>e</i> <sub>5</sub> )	At Birth ( <i>e</i> <sub>0</sub> )	At 1 Yr ( <i>e</i> <sub>1</sub> )	At 5 Yrs ( <i>e</i> <sub>5</sub> )
1955	37.7	50.2	54.9	61.4	66.0	66.8
1956	38.0	50.1	54.3	61.7	66.3	67.0
1957	39.4	52.2	56.3	61.9	66.4	67.1
1958	38.6	51.7	55.9	62.1	66.7	67.4
1959	40.7	52.4	55.8	62.3	66.8	67.5
1960	40.3	52.6	56.5	62.6	66.9	67.7
1961	41.3	52.6	55.9	62.9	67.2	67.8
1962	42.1	53.1	56.7	63.3	67.4	68.0
1963	42.7	53.3	56.9	63.6	67.5	68.1
1964	42.9	52.5	55.6	63.6	67.3	67.9
1965	43.4	53.2	55.8	64.1	67.5	68.1
1966	44.0	52.8	55.8	64.5	67.6	68.1
1967	45.7	53.0	55.6	64.7	67.6	68.1

1973). The diabetes rate was 2.2 times greater; thus, by the beginning of the 1970s, diabetes was already more prevalent among some Indians, such as the Seminoles, than among the general U.S. population (Westfall and Rosenbloom 1971).

The disparity in life-expectancy between Indians and the rest of the U.S. population decreased between 1955 and 1967 (Table 5-9). The increase in life expectancy among Indian people during this time was due primarily to a reduction in the number of deaths among children under the age of five years. When deaths at all ages are examined, the improvement in life expectancy was eight years. But when deaths among those less than one year are excluded, the improvement was only 2.8, and when those under five years are excluded the improvement was only 0.7 years (Hill 1970).

Despite the small gains in life-expectancy for individuals older than five, the oldest Native Americans were living longer. Between 1960 and 1979, mortality rates declined among all Minnesota Indians 75 years of age and older, among males 65 through 74 years of age and among females 25 through 54 years of age. Declines in mortality rates for all causes of death during these decades affected only 8% of males versus 53% of females. No other age groups (14 in all) exhibited declines in mortality. The greatest decline in mortality rates for males and females occurred among those 75 years of age and older (Ringhand, Snowdon, and Johnson 1990).

The top two causes of death among Native Americans, accidents and heart disease, remained consistent from 1955 to 1971 (Table 5-10). Accidents had become the primary cause of death for Native Americans during the middle part of the century (Tables 5-10 and 5-11), a result of a dramatic increase since the 1920s and 1930s, when Townsend (1938) described Native Americans as insulated from the accidents so common in the larger cities and heavily populated areas. Cancer was the fifth leading cause in 1955 and became the third leading cause of death in 1971. The most dramatic increases were seen in cirrhosis of the liver, suicides, and diabetes.

**TABLE 5-10. Changes in crude cause-specific death rates (per 100,000) among American Indians compared with the total U.S. population (IHS 1974; Jackson et al. 1974).**

<b>Cause of Death</b>	<b>1955</b>	<b>1971</b>	<b>Percent Change</b>	<b>Ratio to U.S. Total Pop., 1971</b>
Accidents	155.6	157.1	1	2.9
Heart Disease	133.8	142.0	6	0.4
Cancer	59.1	62.5	6	0.4
Cirrhosis of Liver	14.2	45.6	221	2.9
Stroke	46.4	42.8	-8	0.4
Influenza and Pneumonia	89.8	38.6	-57	1.4
Diseases of Early Infancy	67.6	29.6	-56	1.5
Diabetes	13.9	23.0	65	1.3
Homicide	15.9	20.6	30	2.4
Suicide	8.7	18.7	115	1.7
Congenital Malformations	19.0	10.9	-43	1.5
Tuberculosis	55.1	7.8	-86	3.7
Enteritis	39.2	4.4	-89	4.0

Accidents were a major factor in mortality among Native Americans living in Kansas at the time (Table 5-12). Though considerably high, most likely reflecting the small sample size of Kansas Indian deaths, accidents ranked second in causes of death, behind heart disease. The rate decreased from 1972-1974 to 1975-1977. Deaths due to diabetes saw an increase between the two time periods, perhaps reflecting the rising trend for diabetes indicated in national data for Native Americans in the 1960s, though the overall number of deaths was low.

### **1980s to the 21<sup>st</sup> Century**

The death rate from ischemic heart disease among the Pima increased significantly between 1975 and 1989, ranking third during the first half of the period and first during the second half (Sievers, Nelson and Bennett 1996). Between 1984 and 1988 the cardiovascular

**TABLE 5-11. Changes in crude death rates per 100,000 for Indians and the total U.S. population for selected causes – 1955, 1960, 1965-1967 (Hill and Spector 1971).**

Cause of Death	1955		1960		1965		1966		1967	
	Indian	U.S. Total	Indian	U.S. Total	Indian	U.S. Total	Indian	U.S. Total	Indian	U.S. Total
Accidents	155.6	56.9	155.2	52.3	177.3	55.7	184.1	58	180.9	57.2
Diseases of the heart	133.8	356.5	135.5	369	134.2	367.4	142.3	371.2	140	364.5
Malignant neoplasms	59.1	146.5	65.2	149.2	67.5	153.5	70.9	155.1	70.9	157.2
Influenza and pneumonia excluding newborn	89.8	27.1	95	37.3	64.5	31.9	65	32.5	53.5	28.8
Certain diseases of early infancy	67.6	39	66.7	37.4	53.1	28.6	55.6	26.4	49.4	24.4
Vascular lesions affecting the central nervous system	46.4	106	47.8	108	49.2	103.7	47.5	104.6	48.8	102.2
Cirrhosis of the liver	14.2	10.2	20.7	11.3	29.6	12.8	40.8	13.6	38.9	14.1
Homicide	15.9	4.5	16.1	4.7	19	5.5	14.5	5.9	19.9	6.8
Diabetes mellitus	13.9	15.5	14.3	16.7	20.5	17.1	21.1	17.7	19.4	17.7
Suicide	8.7	10.2	11.4	10.6	12.1	11.1	11.7	10.9	17	10.8
Tuberculosis, all forms	55.1	9.1	23.1	6.1	19.4	4.1	16.7	3.9	16.3	3.5
Gastritis, duodenitis, enteritis colitis, excluding newborns	36	4.7	30.5	4.4	20.7	4.1	19.6	3.9	14.5	3.8
Congenital malformations	19	12.5	19.9	12.2	16.6	10.1	15.8	9.3	13.2	8.8
All other	212.1	131.8	209	135.5	195	137.6	197.5	138.5	181.2	136

**TABLE 5-12. Number of deaths and crude death rates (per 100,000) for Indians of the IHS Kansas Service Unit, 1972-1974, 1975-1977 (IHS 1979).**

Cause	1972-1974		1975-1977	
	Number	Rate	Number	Rate
Diseases of the Heart	17	639.1	16	526
Accidents	8	300.8	6	197.2
Cerebrovascular Diseases	4	150.4	0	0
Influenza and Pneumonia	2	75.2	0	0
Certain Causes of Mortality in Early Infancy	2	75.2	0	0
Diabetes Mellitus	0	0	4	131.5
Cirrhosis of the Liver	0	0	3	98.6
Suicide	0	0	1	32.9
All Other Causes	6	225.5	9	295.9
All Causes	39	1466.2	39	1282.1

mortality rates in thirteen tribes from three different regions (Arizona, Oklahoma, and North and South Dakota) that participated in the Strong Heart Study exceeded the mortality rates found among comparative state and U.S. general population rates (Lee et al. 1998). All-cause mortality among Indian populations in the Strong Heart Study was approximately two times greater than that for the total U.S. population as well as for the U.S. white population. Only for the 65-74 year age group were mortality rates for Indian populations near those for the rest of the U.S. population. Among those between 45 and 64 years of age, Indian mortality rates from cardiovascular diseases were close to the general U.S. rates in the states of Arizona and Oklahoma but were more than double that of the general populations of North and South Dakota. The Strong Heart Study confirmed that cardiovascular disease, diabetes, and cancer had surpassed infectious diseases and injuries as the leading causes of death among American Indians in this age group.

When the 1980s began, cancer in general was less common among Native Americans than whites, particularly among males. Native Americans had significantly lower rates for cancers of the lung, breast, and colon than whites but higher rates for gallbladder, kidney and cervical cancers (Sievers and Fisher 1983). Cancers would become the number two cause of death among Native Americans by the beginning of the 21<sup>st</sup> century, although the percentage of deaths due to cancer would remain lower than that for whites (Anderson 2002).

Prior to 1940, few cases of diabetes were recorded among Native American people. Since then, type 2 diabetes has become a major health concern. By the mid-1980s, diabetes was the seventh leading cause of death among Indians, exceeding by 2.8 times the total U.S. age-adjusted mortality rate. Diabetes had become a major cause of related morbidities and disabilities, because among Native Americans the disease is often diagnosed in the late stages, only after

symptoms become acute. A significant number of Indian people had experienced limb amputations as well as circulatory disorders and kidney failure, all due to diabetes (Campbell 1989). A number of Native Americans suffer from eye problems associated with diabetes. Diabetic retinopathy is a leading cause of blindness, and diabetics are 25 times more likely to suffer from blindness than those without the disease (Gohdes 1995).

Nonetheless life expectancy among Native Americans had increased to 71.1 years by 1980 (Table 5-13)—an increase of nearly 20 years since 1940. Females in particular experienced a marked increase in life expectancy, reflecting a reduction in deaths due to child birth and tuberculosis. Males fared less well, still suffering the consequences of high mortality due to accidents at young ages (Snipp 2006). Native American mortality in the early 21st century is comparable to that of other racial groups in the United States. Table 5-14 shows mortality rates by cause and by race for the year 2000. All groups shared the top two causes of mortality. Native Americans differed in having the highest rankings for accidents (3), diabetes (4), chronic liver diseases and cirrhosis (6) and suicide (8 – tied with Asian/Pacific Islanders). Native Americans had the lowest rankings for strokes (5) and kidney disorders (10).

**TABLE 5-13. Life expectancy at birth among American Indians, 1940 – 1980 (Snipp 2006).**

<b>Year</b>	<b>Both Sexes, Years</b>	<b>Males, Years</b>	<b>Females, Years</b>
1940	51.6	51.3	51.9
1950	60.0	58.1	62.2
1960	61.7	60.0	65.7
1970	65.1	60.7	71.2
1980	71.1	67.1	75.1



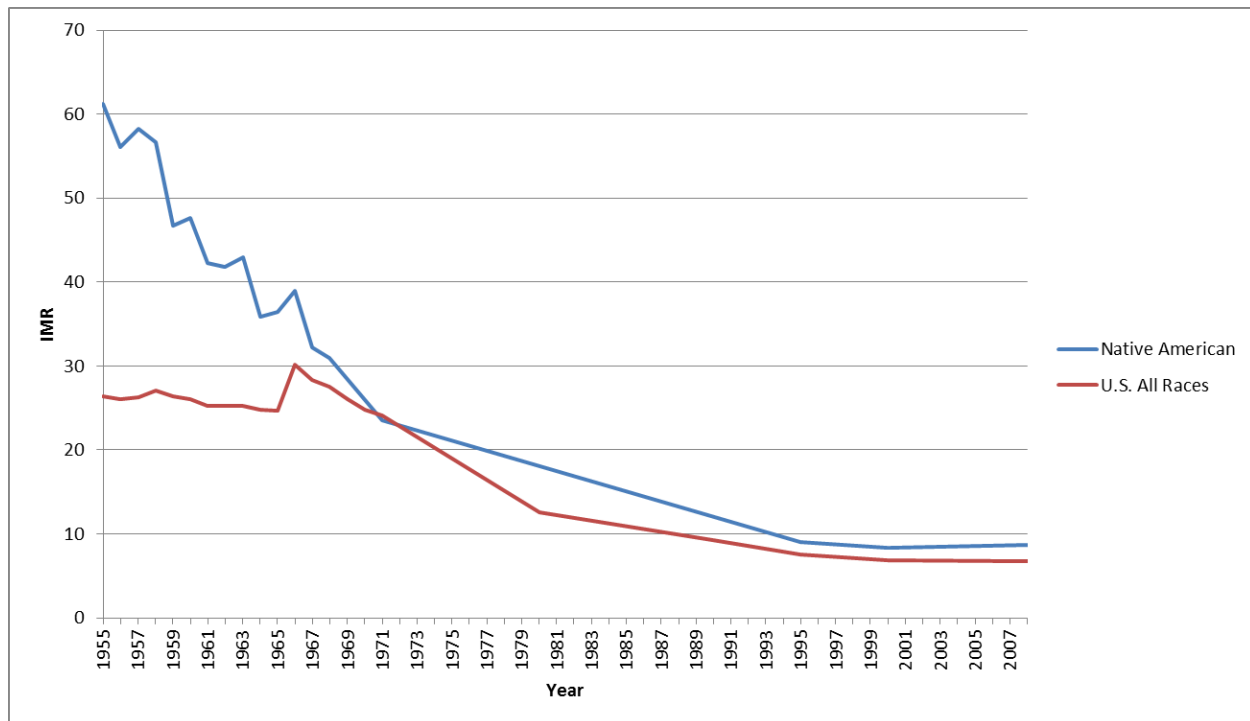
**TABLE 5-14. Causes of Mortality by percent and by race, 2000 (Anderson 2002).**

Cause	White			Black			American Indian			Asian or Pacific Islander		
	rank	#	%	rank	#	%	rank	#	%	rank	#	%
	...	2,071,287	100	...	285,826	100	...	11,363	100	...	34,875	100
Diseases of heart	1	621,719	30	1	77,523	27	1	2,417	21	2	9,101	26
Malignant neoplasms	2	480,011	23	2	61,945	22	2	1,914	17	1	9,221	26
Cerebrovascular diseases	3	144,580	7	3	19,221	6.7	5	572	5	3	3,288	9.4
Chronic lower respiratory diseases	4	112,840	5.4	8	7,607	2.7	7	429	3.8	5	1,133	3.2
Accidents (unintentional injuries)	5	82,592	4	4	12,277	4.3	3	1,353	12	4	1,678	4.8
Influenza and pneumonia	6	57,914	2.8	10	5,990	2.1	9	289	2.5	6	1,120	3.2
Diabetes mellitus	7	55,561	2.7	5	12,021	4.2	4	616	5.4	7	1,103	3.2
Alzheimer's disease	8	46,460	2.2	...	2,728	1	...	95	0.8	...	275	0.8
Nephritis, nephrotic syndrome and nephrosis	9	29,598	1.4	9	6,911	2.4	10	215	1.9	9	527	1.5
Intentional self-harm (suicide)	10	26,475	1.3	...	1,962	0.7	8	297	2.6	8	616	1.8
Chronic liver disease and cirrhosis	...	22,982	1.1	...	2,737	1	6	534	4.7	...	299	0.9
Certain conditions originating in the perinatal period	...	8,532	0.4	...	5,021	1.8	...	124	1.1	10	392	1.1
Assault (homicide)	...	8,339	0.4	6	7,867	2.8	...	203	1.8	...	356	1
Human immunodeficiency virus (HIV) disease	...	6,498	0.3	7	7,848	2.7	...	57	0.5	...	75	0.2

## Discussion

### Health Improvements in Native American populations

The health of Indian people has improved in many ways since the 1950s. Average life expectancy of Indian people has increased by 10 years since 1955, reaching 72.5 years (IHS 2005, 2009; Wolsey and Cheek 1999). During this same period, heart disease became the leading cause of death, at 170 deaths per 100,000 population, whereas half of all IHS and contracting hospital beds were occupied by tuberculosis patients in 1956. Unintentional injury was the next most common cause of death in the mid-1950s, with motor vehicle deaths accounting for half of the total. Bacterial and viral infections of the gastrointestinal tract were prevalent, a result of the lack of indoor plumbing and a safe water source in 80% of Indian homes (IHS 2005). Since the 1950s there has been a significant decline in deaths from tuberculosis, gastroenteritis and other infectious diseases (Young 1996). The rate of accidental



**FIGURE 5-2. Declines in infant mortality since 1955 among Native Americans and the total U.S. (CDC 2012; IHS 1974; NCHS 2005; Wallace 1972).**

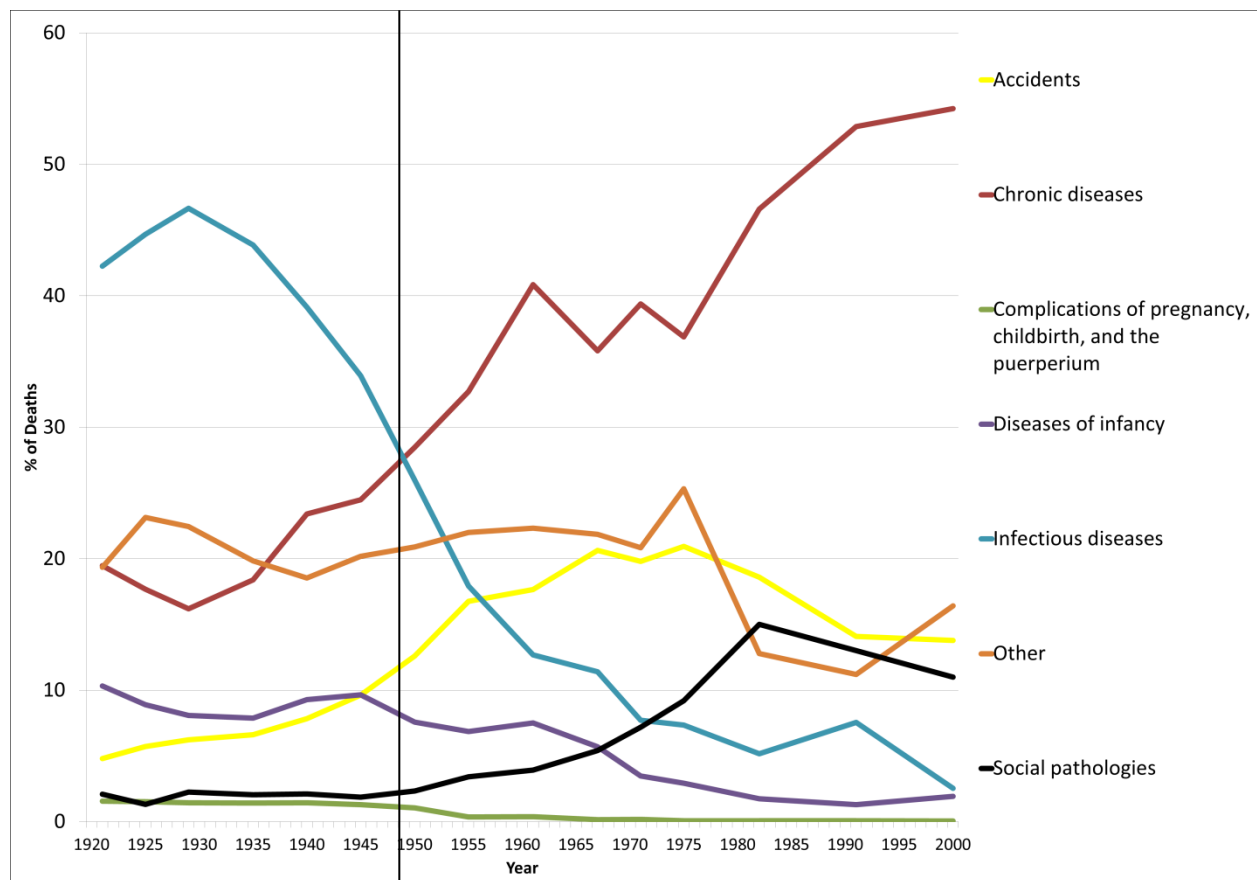
and traumatic injuries among Indians decreased 36% between 1954 and 1998 (IHS 2005) and there has been a steep decline in infant deaths (Rhoades 2003; Young 1996), shown in Figure 5-2.

The termination policy led to the modernization of many Indian people, accompanied by shifts in Indian morbidity and mortality patterns (Tyroler and Patrick 1972), with decreases in deaths caused by infectious diseases and increases in chronic conditions and accidents (Hackenberg and Gallagher 1972; Lang 1985). Termination also led to the creation of the IHS, and federal responsibility for Indian health grew dramatically. Federal appropriations for Indian health services were 155 times greater when the IHS was created than when funds were first appropriated for Indian health in 1911 (Bergman et al. 1999; Campbell 1994).

The passage of the Indian Sanitation Facilities Act (Public Law 86-121) in 1959 allowed the IHS to construct water and sanitary waste facilities for Indian communities. The program has been vital to improving sanitary conditions and reducing communicable diseases due to water contamination (IHS 2005). The act is seen by some as having a more significant effect on Indian health than any effort since smallpox vaccinations were introduced (Bergman et al. 1999).

## Epidemiologic Transition

According to studies on epidemiologic transition in the general U.S. population, as early



**FIGURE 5-3. Mortality transition among Native Americans in the U.S. Created using source data from the years 1921, 1925, 1929, 1935, 1940, 1945, 1950, 1955, 1961, 1967, 1971, 1975, 1982, 1991, and 2000. (Bureau of the Census 1924, 1927, 1932, 1937, 1943; IHS 2004; NCHS 1964, 1974, 1977; NOVS 1947, 1953, 1957).**

as the 1920s cardiovascular diseases had begun to overtake infectious diseases as the primary causes of mortality. These studies attributed the transition to better sanitation, improved water supplies, and public health programs (Levison, Hastings, and Harrison 1981; Omran 1977). The available data suggest that the point at which Native Americans entered the *Age of Degenerative and Man-Made Diseases* occurred around 1949, over 25 years after the transition occurred for the general U.S. population (Figure 5-3). Unfortunately, the Census Bureau did not isolate deaths among Native Americans in its 1949 Vital Statistics of the United States, instead grouping them into a “non-white” category. BIA data are also lacking as this was during the termination period. Census data from 1945 and 1950 specific to Native Americans are available, however, and show significant changes in causes of death for this period (Table 5-15). TB dropped from the first to the third most common cause of death, and heart disease increased from the fourth to the first. Pneumonia and flu and other infectious and parasitic diseases dropped in rank; stroke, cancers and accidents increased.

With the epidemiologic transition came increases in life expectancy. Life expectancy at birth for Native Americans increased dramatically between 1940 and 1990. Male life expectancy increased 17.8 years to 69.1. Among females, life expectancy increased 25.6 years to 77.5 (Young 1996). The primary contributor to the improvement in life expectancy in recent years among Native Americans has been a significant reduction in deaths during childhood (Young 1994). The most significant decrease in mortality has been due to the reduction in tuberculosis and gastroenteritis deaths (Young 1996).

Proposition four of Omran’s (1971) theory of epidemiologic transition explicitly states that the shifts in morbidity and mortality are related to the demographic and socioeconomic transitions that are a part of the process of modernization. Boarding schools, military service,

**TABLE 5-15. Causes of Mortality by percent among Native Americans in 1945 and 1950 (NOVS 1947, 1953).**

Cause	1945	1945 Rank	1950	1950 Rank
Heart disease	9.63	4	13.54	1
Accidents	9.63	5	12.61	2
Tuberculosis	17.60	1	11.51	3
Other	14.75	2	11.37	4
Influenza and Pneumonia	11.48	3	10.92	5
Perinatal Conditions	5.12	7	7.57	6
Other diseases of the digestive system	8.09	6	6.78	7
Cancers	3.76	9	6.13	8
Stroke	3.33	10	4.35	9
Other infectious and parasitic diseases	4.71	8	3.40	10
Alzheimer's disease	1.70	12	2.03	11
Kidney failure	1.59	13	1.24	12
Diabetes	1.40	14	1.08	13
Other diseases of the respiratory system	0.86	17	1.06	14
Homicide	1.06	15	1.04	15
Other diseases of the nervous system	1.02	16	1.01	16
Suicide	0.41	20	0.79	17
Other diseases of the genitourinary system	0.66	18	0.70	18
Liver diseases	0.57	19	0.65	19
Other endocrine, nutritional and metabolic diseases	0.23	22	0.52	20
Other diseases of the nervous system and sense organs	0.18	23	0.47	21
COPD	0.27	21	0.14	22
Septicemia	0.09	24	0.05	23
Hypertensive disease	0.05	25	0.00	24
Other diseases of the circulatory system	2.88	11	0.00	25
All Causes	100		100	

and the termination policy in the postwar years led to modernization among Native Americans (Prucha 1976; Tyroler and Patrick 1972). As services were eliminated on reservations, many Native Americans left the reservations and moved to urban areas for work, where they became part of the market economy and began to purchase mass-produced manufactured goods and food.

Predictable changes in food intake, in particular, are a part of the process of modernization and contribute to the shifts in mortality patterns seen in the epidemiologic transition (Popkin 1993).

Specific factors that have contributed to the shift in disease patterns of Indian people include the reduction of infectious diseases; an abundance of food—particularly those high in sugar, fat, and calories; cigarette smoking; and the adoption of an “American” way of life. As a consequence of these changes, chronic conditions now account for six of the ten leading causes of death among Native Americans, and obesity and diabetes are affecting Indian communities in epidemic proportions (IHS 2005).

The IHS was successful at reducing the infectious diseases so rampant among Native Americans during the first half of the century. With the intervention of this health care system, imposed from outside Indian communities, many infectious diseases were cured pharmacologically and were eliminated relatively quickly as major causes of mortality. Fertility decreased more slowly, leading to a younger population. Behaviorally influenced conditions and deaths increased rapidly with modernization (Broudy and May 1983).

### **Modern Health Characteristics**

A rise in chronic conditions including diabetes, heart disease, cancer, and gallbladder disease is a primary feature of the epidemiologic transition among Native Americans in the last 40 years (Manson and Altschul 2004). However, Native American groups still display a modern disease pattern that is distinct from other ethnic and racial groups in the U.S.

#### *Diabetes*

Few cases of diabetes were documented for Native Americans prior to World War II, but high diabetes mortality among Native Americans began to be observed in the mid-1950s

(Gohdes 1995; Szathmary 1990; Young 1994, 1996). The highest rates of diabetes in the U.S. are now found among Native American tribes, with some having an estimated prevalence of greater than 37%, and some, as high as 50% (Black 2002; Knowler et al. 1993).

From the 1950s through the 1970s, studies began to emerge that documented the rising rates of obesity and diabetes among formerly unaffected populations. West (1974) was among the first epidemiologists to review the newly accumulating literature on diabetes among Native Americans and other indigenous populations throughout the world. Studies conducted in the 1960s and 1970s indicated that Eskimo groups were not susceptible to the diabetes epidemic sweeping through other native North American populations (Scott and Griffith 1957; Mouratoff, Carroll, and Scott 1967; Mouratoff and Scott 1973; Saglid et al. 1966), but in the 1980s, the incidence of diabetes among the Eskimos also increased, although not to the degree seen in other Native Americans (Schraer et al. 1988; Young et al. 1992). Among Cree and Ojibwa populations, diagnosis of diabetes increased dramatically after 1980 (Young et al. 1985). Among the Navajo, the prevalence of obesity and diabetes increased markedly between 1955 and 1988: the prevalence of obesity went from 18.9% to 43.7%, while prevalence of diabetes went from 1.2% to 12.4% (Hall, Hickey, and Young 1992). By the end of the 1980s, Johnson and Taylor (1991) estimated age-adjusted incidence of diabetes among Native American populations to be 11% in males and 13% in females, more than double that of the U.S. population as a whole. By the end of the 20<sup>th</sup> century, diabetes was 230% more prevalent among Native Americans than among the U.S. population as a whole (IHS 1998). More recently the prevalence of self-reported rates of diabetes was 9.7%, compared to 5.7% among non-Indian populations (CDC 2003b).

The prevalence of diabetes varies widely among different Native American tribes (Szathmary 1990). The Akimel O'odham (Pima) Indians of Arizona have the highest incidence

of diabetes of any population studied, and the prevalence of the disease was found to be 19 times higher among the Pima than among a comparative white population. The disease has a strong family component among the Pima and is correlated with obesity (Knowler et al. 1978; Knowler et al. 1983; Knowler et al. 1990). Among Native Americans surveyed from Washington, Oregon, and Idaho, the prevalence of diabetes, adjusted for age, differed greatly among tribes of different culture areas. The highest frequency was found among the Great Basin groups (8.8%), with those of the Plateau having a somewhat lower rate (7.2%). Tribes of the Northwest Coast had the lowest rate of the three groups (4.5%) (Freeman et al. 1989). High diabetes mortality rates have been reported for American Indians in Wisconsin (Reeves et al. 1997), and type 2 diabetes is the leading killer among Native American women in Arizona (Lee et al. 1998).

Using data from multiple sources, Young (1994) was able to identify predictor variables for diabetes among Native American groups that explained nearly half of the total variation. The most important variable was latitude, followed by the region/language groupings Northeast-Algonkian, Northeast-Iroquoian, Subarctic-Algonkian, Plains-Siouan, and Plains-Algonkian. Based on diabetes registry data and the results of glucose tolerance tests, Young constructed three prevalence groups. Among the low-prevalence groups were Eskimos, various Subarctic Athapaskan groups, Aleuts, many Northwest Coast tribes, and the Navajo. The high-prevalence group comprised a number of Southwest tribes including the Pima, Papago, Cocopah, Yuma, and Zuni, as well as the Iroquoian-speaking Cherokee and Seneca. The medium-prevalence group, with a diabetes registry rate between 5% and 15%, and prevalence based on glucose tolerance testing between 10% and 30%, included most of the other Native American tribes (Young 1994). It is not clear if these population differences reflect genetic or environmental effects or both.



Diabetes is associated with a number of acute conditions and long-term complications; which include diabetic retinopathy, nephropathy, peripheral neuropathy and coronary and peripheral vascular diseases; all may lead to premature death (Young 1994). End-stage renal disease deaths due to diabetes have been shown to be six times higher among Native Americans than among Whites (Newman et al. 1990). There also is a suspected link between diabetes and gallbladder disease. Native Americans suffer from higher rates of gallbladder disease, gallbladder cancer and related disorders, and their age of onset for these conditions is younger (Shaffer 2005; Weiss 1990; Young 1994).

The fact that the diabetes epidemic emerged in Native American populations only over the last 50 years indicates that numerous environmental factors are involved (Lappe 1994; Weiss 1990). Familial and dietary relationships with diabetes are well documented. Diabetes has also been associated with environmental toxins. Positive correlations between diabetes and PCBs (polychlorinated biphenyls), DDE (dichlorodiphenyldichloroethylene), and HCB (hexachlorobenzene) have been demonstrated in Native Americans (Codru et al. 2007). Recent studies in mice also suggest a causative relationship between arsenic exposure and the pathogenesis of diabetes (Barrett 2011; Paul et al. 2011). Variation in the microbiota in human gastrointestinal tracts has also been implicated in diabetes and related metabolic diseases (DiBaise et al. 2008).

### *Cardiovascular Disease*

Cardiovascular disease has become the leading cause of death among Native Americans and Alaska Natives (IHS 2004; Lee et al. 1998). In the early part of the 1970s, the heart disease death rate for Native Americans was 21% lower than that of the total U.S. population. By the end of the 20th century, the heart disease death rate was 20% higher for Native Americans (IHS

2004). Recent research suggests that the incidence of heart disease and the proportion of premature deaths due to heart disease are higher among Native Americans than among all other U.S. racial or ethnic categories (CDC 2003a, 2004).

Cardiovascular disease death rates among American Indians were over twice as high in North and South Dakota as in Oklahoma and Arizona for those between the ages of 45 and 64 (Lee et al. 1998). Ischemic heart disease deaths were much higher in both American Indian men and women of Wisconsin aged 45 – 64 years, when compared to whites of comparable age, but were lower in Wisconsin American Indians over 65 years of age (Reeves et al. 1997). Low rates of ischemic heart disease have been documented among the Athabaskan-speaking Navajo and Apache, who moved into the southwestern U.S. from the north 1,000 years ago (Young 1996). The relationship between diabetes and heart disease may be an important factor in the differences in heart disease rates between Indian populations. Strokes had become a leading cause of mortality among Native Americans and Alaska Natives by the beginning of the 21<sup>st</sup> century (IHS 2004); by the end of the 20th century, the death rate due to stroke was 14% higher for Native Americans than for the U.S. population as a whole (IHS 2004). High blood pressure is a major risk factor for strokes, heart disease, and aneurysms, and is a primary cause of chronic kidney failure. Next to African Americans, Native Americans have the highest rates of hypertension of any other ethnic group in the U.S. (Barnes, Adams, and Powell-Griner 2005).

### *Cancer*

Cancer was rare among Indian people fifty years ago (IHS 2005). Now cancers have become a significant contributor to mortality among Native American groups (Lee et al. 1998). The recent overall cancer mortality rate is lower among Native Americans than among the general U.S. public, but between 1990 and 2001, cancer mortality increased by 5% among

Native Americans. During the same time period the cancer mortality rate dropped 6% among the general U.S. population (Espey, Paisano, and Cobb 2005). In 2004 Paltoo and Chu determined that the five most common cancers among Native American males were prostate, lung, colorectal, renal, and gastric, in that order. Among Native American females the five most common cancers were breast, colorectal, lung, endometrial, and ovarian. For cancers of the gallbladder, liver, intrahepatic bile duct, and kidneys, Native Americans had higher rates than whites.

Cancer among Canadian First Nations shows a similar pattern as that of Native Americans of the U.S. The incidence of cancer has been significantly lower for First Nations people compared to all Canadians, but cancer rates have been increasing dramatically over the last 40 years, especially cancers of the lung, breast, prostate, and colon (Marrett and Chaudhry 2003). Canadian First Nations females living in Manitoba and Ontario have higher incidence and mortality for cervical and gallbladder cancers than the general population (Marrett and Chaudhry 2003; Rosenberg and Martel 1998). Kidney cancer rates are higher among both sexes of First Nations people. Lung cancer is the number one cause of cancer deaths among First Nations groups and has been increasing dramatically among the women (Rosenberg and Martel 1998). Adoption of a modern lifestyle and heightened exposure to carcinogens very likely play a role in the rise of cancer rates.

### *Social Pathologies*

Mental health problems and social pathologies affect Indians to a high degree and appear to be increasing. Suicides among Native Americans were 85% more common than among the rest of the U.S. near the end of the 20<sup>th</sup> century (IHS 1998). Suicides among Native Americans

grew to be 91% greater than among the general U.S. population and became the eighth most common cause of mortality by the early 2000s (Anderson 2002; IHS 2004).

Commercial cigarette smoking is higher among most Native American groups. Self-reported cigarette smoking was 32.2% (CDC 2003b) and higher than the smoking rate of the U.S. as a whole. Smoking rates are especially high among young Native Americans, and in some age groups are higher among females than among males (Young 1994). Smoking is most common among Indian populations of the Great Lakes and Great Plains regions, at 44% and 42%, respectively (Rith-Najarian et al. 2002).

Deaths due to chronic liver disease, cirrhosis, and other alcohol-related causes such as accidents are seven times the national rate (Berry et al. 2004; IHS 2004; Lee et al. 1998). High rates for liver disease and cirrhosis are attributable to greater consumption of alcohol (Singh and Hoyert 2000). The rate of mortality due to alcoholism among Native Americans has been reported to be as much as 630% higher than the total U.S. rate (IHS 1998) and is significantly higher across age- and sex-specific groups (Reeves et al. 1997).

Injuries from unintentional accidents, suicides, and homicides are responsible for a much greater percentage of deaths among Native American than among Whites (Lee et al. 1998; Young 1996). The increase reflects a rise in the number of deaths due to automobile accidents, the primary contributor to accidental deaths. This dramatic rise in accidents may have caught the IHS off-guard, for this was not generally a topic given much discussion in early IHS literature. Perrot and West's (1957) review of health services for Indians cited the excess mortality due to accidents but concentrated primarily on the containment of infectious diseases. Guyon's (1973) review of challenges to the IHS recognized higher mortality from accidents among Native Americans but otherwise did not discuss the causes or contributing factors. Also, the relocation

policy that grew out of termination urbanized many Native Americans, certainly exposing them to the accidents from which they had been previously insulated (Prucha 1984a).

## **Causes of the Observed Pattern**

### *Obesity*

Obesity is among the leading causes of preventable death in the US (Barness, Opitz, and Gilbert-Barness 2007), and many chronic diseases currently affecting Native Americans are linked to obesity. Heart disease, high blood pressure, diabetes, gallbladder problems and some cancers are strongly correlated with being overweight or obese. In fact, obesity is considered to be among the most significant risk factors for type 2 diabetes, and Native American tribes showing high rates of diabetes also have high rates of obesity (Shaffer 2005; Young 1994).

Body Mass Index (BMI), a measure of weight-for-height, is used to determine an individual's weight status and related risk for chronic degenerative diseases. BMI of 25 or more is an indicator of overweight and BMI over 30, of obesity. These categories are associated with greater risk for a number of diseases. The manner in which fat is distributed throughout the body also plays a role in disease risk. Trunk fat shows more metabolic activity than that located in the thighs, buttocks, and limbs. Obesity of the abdominal region has been recognized as a significant correlate with several chronic conditions, including the development of insulin resistance and associated pathologies (Ivy 1997). Waist circumference and the waist-to-hip ratio have become important measures for identifying abdominal obesity and those at risk (Young 1994). Additionally, obesity has been cited in leptin resistance, interfering with the physiological regulation of appetite in relation to energy intake, further exacerbating the condition (Enriori et al. 2006).

Obesity among American Indians is much more common now than it was 50 years ago (Manson and Altschul 2004): in the 1940s energy and nutrient deficiencies were prevalent among many Native American tribes. In contrast, a 1987 Survey of American Indians and Alaska Natives (SAIAN) found that the proportion of overweight and obese Native Americans exceeded that of all other races and age and sex groupings. The incidence of overweight was 34 percent among Indian males and 40 percent among Indian females, whereas the rates among the rest of the U.S. were 24 percent for males and 25 percent for females (Young 1994). From 1997 to 2000, the Centers for Disease Control data documented self-reported incidence of obesity among 23.9% of Native Americans (CDC 2003b). Abdominal obesity was especially prevalent among the Southern Cheyenne in 1994, with 84% of males and 96% of females affected (Löhn 1995). Native American women of the Southwest have been reported to have a greater tendency than other racial or ethnic groups to accumulate fat around the abdomen rather than on the buttocks and thighs, suggesting an innate pattern of fat distribution among Native Americans places them at higher risk for chronic conditions (Thomas, Keller, and Holbert 1997).

### *Diet*

Research on traditional Native American foods indicates that diets overall included a wide variety of animal and plant species (Kuhnlein and Receveur 1996). The traditional foods of Native Americans consisted of fish and wild animals, wild plants, and cultivated crops. Diet was based on environmental resources and varied considerably among Native Americans across North America. Agricultural groups produced the complementary crops of corn, beans, and squash, as well as other cultivated foods. Wild plants were widely available to all groups except those at the northernmost latitudes. Wild rice, pinyon nuts, acorns, and other naturally abundant foods were major food resources for some populations (Figure 5-4). Buffalo, deer, and



**FIGURE 5-4. Traditional wild rice harvest using birch bark canoes. (Courtesy Minnesota Historical Society)**

pronghorn sheep were regularly hunted by many groups, in addition to numerous smaller wild animals. These wild and cultivated foods provided a nutritionally complete diet for most Native American groups prior to European contact (Berzok 2005; Young 1994).

Native diets are superior to modern Western diets in their lower levels of saturated fat and sodium and higher levels of minerals and fiber (Wirsing et al. 1985). Nutrient comparisons between traditional and modern diets also indicate higher values for protein, iron, magnesium, copper, and zinc in the traditional diet. Meat from domesticated livestock typically has higher percentages of fat and saturated fat, and lower levels of protein. In contrast, wild game supplied plenty of high quality protein as well as fat; furthermore, the fat from wild game is

approximately 38 percent saturated, 32 percent monounsaturated, and 30 percent polyunsaturated fat; and the meat contains predominantly monounsaturated and polyunsaturated fats (Cordain et al. 2005; Eaton, Shostak, and Konner 1988). Fish and fish oils in traditional diets provided regular intake of omega-3 fatty acids, whereas deficiencies of omega-3 are implicated in several chronic diseases associated with modern diets (Kuhnlein and Receveur 1996).

During the reservation period, however, the U.S. government provided food to Indians who were no longer allowed to practice their traditional subsistence patterns. These foods were new to the Indians and generally inadequate nutritionally (de Cora 2001). Flour, lard, bacon, sugar, syrup, and other canned and processed foods became dietary staples. The quantity of food supplied was often insufficient and starvation was a stark reality. Energy and nutrient deficiencies were common occurrences among many Native American tribes into the 1940s. Research among Canadian Subarctic tribes in the 1940s indicated that these groups were often in danger of starvation (Young 1994). Indian students living at boarding schools were fed a diet consisting largely of starchy foods and meat. The typical meal featured baked bread and a stew or meat and gravy. Fresh fruits and vegetables were rarely available, although some schools tried to plant gardens. A few schools with productive school farms could meet the stipulated policy of providing food in abundance. More often, children at Indian boarding schools were underweight and malnourished (Adams 1995; Child 1998).

Assimilation included changes in diet, under the assumption that the modern U.S. diet was superior. Health officials of the time believed that a proper diet could prevent illness, and that dairy products in particular facilitated recovery from tuberculosis (Keller 2002). Milk consumption was encouraged among Indians. Boarding schools such as Haskell and Flandreau ran dairies, although the students were often more likely to consume coffee than milk. Some



school dairies produced a pint per student per day, but most of the milk was turned into butter: students did not actually drink a pint of milk daily. At the time, officials were unaware that most Native Americans were lactose intolerant, and that any significant consumption of milk could lead to diarrhea and nutrient malabsorption among the children (Beers 2003; Beers and Berkow 1999; Flatz 1987).

A shift from traditional subsistence livelihoods to a cash economy began in the early 1900s. As tribal members left the reservations in search of paid employment, the amounts of wild and homegrown foods in the Indian diet diminished, with a greater proportion of food coming from processed and purchased sources (Halpern 2007; Michel, 2004). Additionally, boarding schools introduced desserts and sweets to children. Military service introduced Indian soldiers to fried foods, white bread, luncheon meats, cakes, and breakfast cereals. Both schools and the military introduced Indians to the habit of eating three meals a day. The boarding school students and former World War II servicemen took these eating habits back with them to the reservations. Soon traditional foods were eaten by a minority of tribal members. These dietary changes accelerated greatly after 1940 (Kane and Kane 1972; Löhn 1995; Smith-Morris 2004).

The shift to the new diet was well under way by the mid-1950s. Indian families became more dependent on market and commercially prepared foods. The new consumption pattern featured processed, high-fat meats such as bologna, hot dogs and hamburgers, as well as highly processed starches and sweetened beverages. Consumption of fresh fruits and vegetables became less frequent with fewer gardens and opportunities to gather wild plants (Conti 2006).

Currently, many thousands of American Indian families are dependent on the Food Distribution Program on Indian Reservations (FDPIR). Not surprisingly, these commodities are inexpensive foods, laden with sugar, low in protein and unsaturated fats, and high in saturated

and trans fats. Indian people who take advantage of the commodities program can choose among approximately 80 different foods, but the choices made are often for white flour, lard, processed cheese, and sugary and salty foods to which they became accustomed during the reservation and assimilation periods. Many question whether the program is really helping the people in the long run (Mihesuah 2003; Morell and Enig 2001).

The modern Western diet is higher in calcium and vitamin A but also in total fat and sodium (Kuhnlein and Receveur 1996). The Western diet has been implicated in the rise of chronic diseases not only among American Indians but also in the general U.S. population (Cordain et al 2005). Diets high in processed carbohydrates, fat, and sodium have replaced traditional diets based on a variety of fruits and vegetables, high in complex carbohydrates and fiber and lower in fat (IHS 2001). The Canadian Subarctic tribes that were at risk of starvation in the 1940s showed significantly heavier weights compared to other Canadians by the 1970s (Young 1994). The Southern Cheyenne exhibited extremely high rates of diabetes and obesity by 1994 (Löhn 1995). Today, Native Americans tend toward overconsumption of total calories with disproportionately high energy intake from fat and carbohydrates. Modern fat intake among Native Americans is well above the 30% of total calories prescribed by the recommended dietary allowances (RDA), varying from 31% to 47% (Story et al. 2000).

Demographic variables affect dietary patterns. According to unpublished information gathered as a part of the IHS Diabetes Prevention Program, urban versus rural residence significantly affects food choices, with urban residents consuming healthier foods more often, reflecting food availability, variety, and consumer choices. Age, gender, education level, employment, marital status, and income also affect food choices. Older individuals with higher income and education levels chose healthy foods most often. Younger males with lower income

and less education tend to consume unhealthy foods most often. Native Americans have an economic incentive to purchase less expensive and more calorie-dense, high-carbohydrate foods (Richards and Patterson 2006; Story et al. 2000).

### *Activity*

Changing activity levels are also significant in the health problems currently being experienced by Native people. Physical activity can prevent obesity and associated health problems, but in the process of assimilation, Native Americans have shifted from a traditional subsistence involving greater activity levels to a sedentary lifestyle with decreased physical activity (Halpern 2007). The concomitant shifts in activity patterns and dietary intake most certainly affected energy balance of Native American individuals as they modernized. The dietary changes described above, emphasizing high fat, processed carbohydrates, and more total calories, combined with a reduction in regular activity, served as a dual assault on the metabolism of Native Americans in a relatively short period of time. The relative contributions of diet and inactivity to insulin resistance are not known, but studies do suggest that diet is the predominant factor in obesity, with physical activity playing a lesser role (Bleich et al. 2008). However, activity has a direct impact on the body's ability to regulate glucose and thus independently contributes to metabolic syndrome. As with dietary intake among tribes such as the Southern Cheyenne, activity levels changed dramatically starting in 1940 (Löhn 1995).

Physical inactivity has been cited as a major contributor to insulin resistance (Bloomgarden 1998). Epidemiological studies suggest that people who are physically active are less likely to develop impaired glucose tolerance and type 2 diabetes. The protective effect of physical activity appears strongest among people who are most at risk and is independent of age. Even older people who engage in vigorous activity on a regular basis show greater glucose

tolerance and reduced insulin response to a glucose surge than sedentary people of comparable weight and age (Ivy 1997). Even moderate activity has been associated with significantly higher insulin sensitivity (Mayer-Davis et al. 1998). Estimates suggest that an increase in physical activity of only 500 kcal per week can result in as much as a 6% reduction in the risk of developing diabetes mellitus (Helmrich et al. 1991). Vigorous activity mobilizes abdominal fat and contributes to preventing or lessening insulin resistance associated with central obesity. Physical activity also affects muscle metabolism. Regular activity prevents muscle atrophy and encourages muscle development, which has been found, first, to significantly reduce the insulin response to a glucose load without impairing glucose tolerance and, second, to increase glucose clearance. A loss of muscle mass has been associated with the development of insulin resistance and associated conditions (Ivy 1997).

In addition to burning calories, physical activity may lead to the conversion of glycolytic IIb muscle fibers to oxidative IIa fibers, as well as to an increase in capillary density. The oxidative IIa fibers have a higher capillary density and are more responsive to insulin than IIb fibers. These morphological changes in muscle tissue, in particular in capillary density, are associated with fasting insulin levels and glucose tolerance (Ivy 1997).

### *Genetics*

The relative roles of genetics and environment on body weight and associated conditions is complicated and a subject of marked disagreement among researchers (Halpern 2007). Some studies suggest that the environment is responsible for weight differences among populations of similar genetic background living in different environments, whereas genetics explains differences in weight within populations living in similar environmental circumstances (Ravussin 1995). Klimentidis, Miller, and Shriver (2009) were able to show a genetic relationship between

percent body fat and BMI among Hispanic and Native American populations with European admixture. The FTO gene has been implicated in obesity through an apparent impact on satiety and caloric intake (Speakman, Rance, and Johnstone 2008; Thorleifsson et al. 2009; Willer et al. 2009). However, studies of the genetic determinants of body weight and BMI indicate that genetic variants explain only a small percentage of the overall variation in weight. With regard to fat patterning, genes appear to have a more dramatic effect, particularly with regard to differences in body composition between sexes (Heid et al. 2010; Massachusetts General Hospital 2010). Given greater genetic control of body fat distribution and known associations between abdominal obesity and several chronic conditions (Young 1994), a genetic predisposition for obesity-related conditions at least must be considered.

The genetic cause of diabetes has been widely debated (Szathmary 1990). Wareharn (2004) cites numerous studies that suggest a genetic–environment interaction leads to the observed frequencies of type 2 diabetes among the world’s populations. Native American tribes are biologically and genetically variable, and it is likely that the genetic predisposition for diabetes varies greatly within and between American Indian nations, although the degree of Indian ancestry has been identified as a risk factor for diabetes, independent of obesity (Quiggins 1990; Young 1994). Among the Pima for instance, even those with a healthy BMI still show an eight times greater incidence of diabetes than whites (Knowler et al. 1981). Native Americans having low levels of genetic admixture with non-natives are at higher risk for diabetes, again indicating a genetic connection (Caballero 2005; Gohdes 1995). Among the Eastern Cherokee, diabetes has been found to be most prevalent among those individuals who have a 75% or greater degree of Indian ancestry (Quiggins 1990).

The Strong Heart Family Study found that among 950 American Indians in Arizona, Oklahoma, and the Dakotas there may be separate genes operating in diabetic and nondiabetic patients for obesity and risk factors related to lipid metabolism (North et al. 2003). Researchers have identified three metabolic predictors of obesity among the Pima Indians: low metabolic rates; high 24-hour respiratory quotient; and insulin insensitivity (Halpern 2007; Ravussin, 1995; Swinburn et al, 1991, Walston et al, 1995; Zurlø et al, 1990), although other studies found little difference in metabolic rate between Indians and whites (Story et al. 2003). Weiss et al. (1989) found that diabetes and hyperinsulinemia among Indians could not be explained entirely by obesity and suggested a genetic basis for insulin resistance. Szathmary (1994b) identified a number of possible genetic links to diabetes through glucose control and insulin action. Indeed genes have been identified that may predispose some individuals to diabetes. Walston et al. (1995) identified a mutation in the  $\beta_3$ -adrenergic-receptor gene. Pima Indians homozygous for the Trp64Arg  $\beta_3$ -adrenergic-receptor mutation experience an earlier inception for type 2 diabetes and tend to have a lower resting metabolic rate (RMR). It is hypothesized that the mutation accelerates the inception of diabetes by changing the metabolism in abdominal adipose tissue, as  $\beta_3$ -adrenergic-receptors normally act on the process of lipolysis.

More recent genome wide association studies (GWAS) have identified a number of genetic loci associated with increased risk for diabetes and associated conditions (Grarup, Sparsø, and Hansen 2010). These loci are presented in Table 5-16, including the single-nucleotide polymorphisms (SNP) responsible for the variation. Five in particular are associated with insulin resistance: PPARG, ADAMTS9, GCKR, IRS1, and IGF1. FTO is also associated with insulin resistance but appears to operate indirectly through its effect on obesity. A significant number of the identified genes affect  $\beta$ -cell function in the pancreas. Some affect

**TABLE 5-16. Gene regions with loci associated with diabetes and metabolic syndrome and allele frequencies for selected populations (Grarup, Sparsø, and Hansen 2010).**

Gene/ Region	Nucleotide	SNP	Allele	European	East Asian	West African	Native American	T2D p	MS p
KCNJ11	(C/T)	(rs5215)	T	0.592	0.678	0.992	0.700	0.512	0.244
LEPR	(A/G)	(rs1137100)	A	0.658	0.144	0.883	0.514	0.355	0.268
PPARG	(A/C)	(rs17793693)	A	0.117	0.000	0.008	0.471	0.253	0.045
HHEX	(C/T)	(rs5015480)	C	0.573	0.232	0.547	0.143	0.701	0.032
TCF7L2	(C/T)	(rs7903146)	T	0.250	0.023	0.292	0.129	0.001	0.152
TCF7L2	(G/T)	(rs12255372)	T	0.217	0.023	0.267	0.014	0.002	0.214

SNP = single-nucleotide polymorphisms

T2D p = probability of type 2 diabetes

MS p = probability of metabolic syndrome

incretins, a group of gastrointestinal hormones that promote an increase in the insulin release from pancreatic beta cells after food is consumed but before blood glucose levels can become elevated.

For the ABCC8/KCNJ11 genes at the E31, E33, and E23K loci, haplotypes bearing the A-T-G sequence are positively associated with type 2 diabetes, and haplotypes bearing the G-G-A sequence at the three loci are inversely associated with diabetes. The haplotype associated with a greater risk of diabetes (A-T-G) has a higher frequency in Native Americans than in Europeans, at nearly 50% versus 28% (Parra et al. 2004). Other gene regions with loci associated with diabetes and metabolic syndrome for which the gene frequencies are known for Native American populations are shown in Table 5-16. Of particular note is the SNP at the PPARG gene, a gene known to regulate fatty acid storage and glucose metabolism. The variant allele has a much higher frequency among Native American populations and has been identified as contributing to insulin resistance.

An overview of the physiologic impact of loci associated with diabetes-related traits and of genome wide significance is presented in Table 5-17. Recent genetic studies, however, have uncovered only marginal effects of SNP variation on diabetes (Hanson et al. 2007; Ma et al.

**TABLE 5-17. Overview of the physiologic impact of loci associated with diabetes-related traits at genome-wide significance (Grarup, Sparsø, and Hansen 2010).**

<b>Gene(s)/ Region</b>	<b>Trait</b>	<b>Physiologic Phenotypes</b>	<b>Mechanism</b>
KCNJ11; ABCC8	T2D	Reduced insulin release during OGTT; Increased glucagon levels during hyperglycemic clamp	Impaired $\beta$ -cell function and impaired glucagon suppression
TCF7L2	T2D, FG, 2 h-G	Impaired conversion of proinsulin to insulin; Reduced insulin release during OGTT; Reduced incretin effect; Reduced glucagon levels	Impaired incretin-stimulated insulin release; Impaired expression of prohormone convertases
WFS1	T2D	Wolfram syndrome; Reduced insulin release during OGTT; Reduced GLP-1 induced insulin release during hyperglycemic clamps	Impaired incretin-stimulated insulin release
HHEX; IDE	T2D	Reduced insulin release during OGTT; Borderline significant reduced insulin release during IV glucose stimulation; Reduced birth weight	$\beta$ -cell dysfunction
SLC30A8	T2D, FG	Impaired conversion of proinsulin to insulin; Reduced insulin release during OGTT; Reduced insulin release during IVGTT	Impaired formation of insulin granules impairing insulin release
CDKAL1	T2D	Impaired conversion of proinsulin to insulin; Reduced insulin release during OGTT; Reduced insulin release after IV glucose stimulation; Reduced birth weight	$\beta$ -cell dysfunction
CDKN2A; CDKN2B	T2D	Reduced insulin release during OGTT; Reduced insulin release during IVGTT	$\beta$ -cell dysfunction
IGF2BP2	T2D	Reduced insulin release during OGTT; Reduced insulin release during IVGTT; Reduced insulin release after IV tolbutamide stimulation	$\beta$ -cell dysfunction
KCNQ1	T2D	Reduced insulin release during OGTT; Reduced glucose-stimulated incretin secretion	Decreased incretin secretion
JAZF1	T2D	Reduced insulin release derived from OGTT	Possibly $\beta$ -cell dysfunction
CDC123; CAMK1D	T2D	Reduced insulin release during OGTT; Reduced arginine-stimulated and second-phase glucose- stimulated insulin release during hyperglycemic clamp	Reduced $\beta$ -cell mass
THADA	T2D	Reduced GLP-1—and arginine- stimulated insulin release during hyperglycemic clamp	Reduced $\beta$ -cell mass due to increased apoptosis
MTNR1B	FG, T2D	Reduced insulin release during OGTT and IVGTT	Impaired melatonin-stimulated insulin release



**TABLE 5-17 (continued). Overview of the physiologic impact of loci associated with diabetes-related traits at genome-wide significance (Grarup, Sparsø, and Hansen 2010).**

<b>Gene(s)/ Region</b>	<b>Trait</b>	<b>Physiologic Phenotypes</b>	<b>Mechanism</b>
MADD	FG	Higher fasting proinsulin	Insulin processing defect
ADRA2A	FG	Reduced insulin release during OGTT	$\beta$ -cell dysfunction
TSPAN8	T2D	Reduced insulin release during OGTT	Possibly $\beta$ -cell dysfunction
FADS1	FG	Reduced insulin release during OGTT	$\beta$ -cell dysfunction
GLIS3	FG	Reduced insulin release during OGTT	Possibly $\beta$ -cell dysfunction
C2CD4B	FG	Reduced insulin release during OGTT; Increased fasting proinsulin	Impaired insulin processing and release
PROX1	FG, T2D	Reduced insulin release during OGTT	$\beta$ -cell dysfunction
GCK	FG, T2D	Increased glucose levels during OGTT; Reduced insulin release derived from OGTT	Increased glucostatic set point and impaired $\beta$ -cell function
DGKB; TMEM195	FG, T2D	Reduced insulin release during OGTT	$\beta$ -cell dysfunction
G6PC2	FG	Increased insulin release during OGTT; Increased insulin release during IVGTT; Increased basal hepatic glucose production; Decreased risk of T2D	Unknown
GIPR	2 h-G	Reduced insulin release during OGTT; Increased fasting proinsulin; Impaired incretin effect	Impaired incretin-stimulated insulin release ; Impaired insulin processing
CENTD2	T2D	Lower HOMA-B	Impaired $\beta$ -cell function
PPARG	T2D	Decreased insulin sensitivity	Whole-body insulin resistance
ADAMTS9	T2D	Reduced insulin-stimulated glucose uptake during hyperinsulinemic-euglycemic clamp; Increased glucose-stimulated insulin release	Peripheral insulin resistance
GCKR	FG, T2D, 2 h-G, triglyceride	Increased insulin resistance derived from fasting and OGTT; Increased insulin-stimulated hepatic glucose output	Hepatic insulin resistance
IRS1	T2D	Increased insulin resistance derived from fasting and OGTT	Whole-body insulin resistance
IGF1	FI	Increased HOMA-IR	Whole-body insulin resistance
FTO	BMI, T2D	BMI-dependent insulin resistance	Increased BMI-dependent insulin resistance
HNF1B	T2D		Unknown
ADCY5	FG, 2 h-G, T2D, birth weight	Decreased birth weight	Unknown
RBMS1	T2D	Increased HOMA-IR	Unknown
KLF14	T2D	Increased fasting insulin and HOMA-IR	Unknown

2008). More GWAS studies on risk susceptibility variants and gene frequency studies need to be completed on a larger number of Native American populations before a genetic basis for high

rates of diabetes among Native Americans can be confirmed and Neel's (1982) thrifty genotype hypothesis supported unequivocally.

Genetics might provide an explanation for the observed differences in rates of diabetes among populations and individuals, but it is not a sufficient explanation for the dramatic changes in disease prevalence observed in some populations in the last few decades. There has not been sufficient time for genetic change in Native American populations to account for the transition from low to dramatically high prevalence of diabetes. Additionally, there is strong evidence that the rise in chronic disease among Native Americans is related to environmental factors such as dietary intake and activity patterns (Kunitz 2004; Lappe 1994; Szathmary 1994a; Young 1994; Yach, Stuckler, and Brownwell 2006). A strictly genetic explanation for the rise of type 2 diabetes has met with other challenges. The evolutionary basis of diabetes is contested by the fact that seasonal or sporadic periods of nutritional deficiencies may not be a major factor on the health of traditional societies (Wirsing et al. 1985).

Genetics has been implicated in other diseases disproportionately affecting Native Americans. Alcoholism, gallbladder disease, and some cancers appear to be more common among people of Native America heritage. Weiss et al. (1984) postulated a genetic component to Native American susceptibility to gallbladder disease, noting the disease pattern among Native Americans did not mirror that predicted solely by the process of Westernization. The high prevalence and geographic distribution of gallbladder diseases in Native Americans suggests a genetic component, and their association with obesity and diabetes may suggest a common genetic origin as part of the New World syndrome (Weiss, Ferrell, and Hanis 1984; Weiss et al. 1984). Among non-Indian populations, gallbladder disease is associated with ischemic heart

disease and cancers of the colon, rectum, prostate, breast and endometrium—conditions that affect Native Americans at much lower rates.

Cancer mortality in populations of mixed Native American ancestry is intermediate between that for the general U.S. population and that of tribes with higher degrees of full-blood Native American ancestry. Tribes in Oklahoma as well as Hispanics—generally showing Native American and European admixture—have intermediate cancer rates compared with less admixed populations. For instance, in the early 1980s lung cancer was nine times more prevalent among Native Americans in Oklahoma than in the Southwest, yet the rates of the Oklahoma groups still were lower than in non-Indian populations. Moreover the types of cancers experienced by Native Americans differ by organ, with gallbladder, kidney, and cervical cancers being more prevalent (Sievers and Fisher 1983). This may suggest a genetic component to cancer risk.

Genetic differences in the enzymes of alcohol metabolism have been documented for Native Americans and are implicated in the higher rates of alcoholism (Mulligan et al. 2003; Reed 1985). Differences in enzyme production however do not adequately account for the high rates of alcohol use nor the associated morbidity and mortality experienced by Native Americans (Hackenberg and Gallagher 1972; Kunitz 2004; Young 1996). Other non-Native American populations exhibit similar deficiencies without showing the associated social pathologies (NIAAA 2002).

Many studies indicate a complex interaction between genes and environmental factors in the etiology of type 2 diabetes (Szathmary 1994b). A lower prevalence of diabetes mellitus and obesity among the Pima living in Mexico than those living in the U.S. indicates that even when populations are genetically predisposed to develop these conditions, their expression is determined largely by environmental conditions. This is strong evidence that lifestyle changes

associated with acculturation contribute most significantly to the prevalence of type 2 diabetes in a population (Schultz et al. 2006).

Environmental and lifestyle factors could impact the expression of genes through epigenetic processes. The dramatic increase in weight seen among the Canadian Subarctic tribes between the 1940s and 1970s for instance could reflect epigenetic changes in response to the near starvation conditions experienced by the population in the 1940s. Given the abrupt onset of chronic conditions among Native Americans, epigenetic explanations may be more valid. Genetic adaptations proceed slowly in populations, whereas epigenetic adaptations may occur in a single generation or across just a few generations (Gluckman et al. 2008; Gluckman, Hanson, and Spencer 2005; Kuzawa and Thayer 2011).

### *Economic Status*

Native Americans in general, whose annual income was only one-fifth that of whites in 1970 (Wallace 1972), remained the poorest demographic group into the 1970s (Table 5-18). Disparities in socioeconomic status very likely contributed to many of the health disparities recorded. Studies suggest that poverty and unemployment are closely related to health disparities (Castor et al. 2006), alcohol consumption (Singh and Hoyert 2000), cigarette smoking (Flint and Novotny 1997) and other social pathologies (Trovato 1988).

The relationship between poverty and obesity also is well documented. The highest obesity rates occur among populations with the highest poverty rates and lowest education levels. High poverty and unemployment limit the ability of many Indian people to purchase healthier foods and encourage reliance on cheap, energy-dense foods and the special federal commodity programs for tribes (Halpern 2007; PRC 2004). An inverse relationship exists between the caloric density of foods and their cost: foods made of refined carbohydrates with added sugar

**TABLE 5-18. Annual income of selected racial groups in 1970 (Wallace 1972).**

<b>Race</b>	<b>Annual Income, 1970</b>
White	\$10,240.00
Hispanic	\$7,330.00
Black	\$6,280.00
American Indian	\$1,900.00
All Groups	\$9,870.00

and fat tend to be the lowest in consumer cost. Food and income insecurity are associated with reduced expenditures on food, lower consumption of fruits and vegetables, and less nutritional diets (Drewnowski and Specter 2004). In recent years, the “food desert” concept has developed as an explanation for disparities in obesity. The concept suggests that racial, socioeconomic, and geographic variations in obesity prevalence in the U.S. are the result of inequalities in the nature of the retail food system. Substandard retail food services—featuring a concentration of fast food restaurants and convenience stores and a lack of large supermarkets in low SES areas—in conjunction with limited individual and familial economic assets, contribute to the increased risk of obesity among poor and minority populations (Ford and Dzewaltowski 2008).

Socially or economically disadvantaged groups are also at higher risk for developing diabetes (Knowler, McCance, Nagi et al. 1993). Diabetic adults are more than twice as likely to have less than a high school education compared to the nondiabetic population. Diabetics tend to earn less, with a median individual yearly income less than half that of nondiabetics, and diabetics are more likely to be unemployed. Socioeconomic inequities may act directly on the risk factors for diabetes, including poor dietary intake, obesity, access to health care, and behavioral risk factors such as cigarette smoking, alcohol consumption and stress (Black 2002; Lynch, Kaplan, and Shema 1997; Robbins et al. 2001). Additionally, low socioeconomic status may contribute to the development of diabetes due to inadequate maternal dietary intake leading

up to and during pregnancy (Barker 1994). Recently, Ludwig et al. (2011) demonstrated that people in poverty-stricken neighborhoods had higher rates of obesity and diabetes, but that individuals who moved from a neighborhood with a lower poverty level could reduce their risk of obesity and diabetes.

### *Historical Trauma and Structural Violence*

Native Americans experienced an immense disruption of their lives, land base, and culture following contact with Europeans. Colonization produced a lasting legacy of trauma and grief over many generations. This historical trauma and unresolved grief contribute to the current social pathologies seen in high rates of homicide, suicide, child abuse, domestic violence, substance abuse, and mental health problems experienced by Native Americans (Brave Heart and DeBruyn 1998).

Historical trauma and loss are often cited as factors in the poor psychological and physical health of Indians, contributing to the observed health disparities between them and other populations. Historical traumas are the collection of psychological injuries that occur during the life span as well as across generations due to the difficulties Native Americans experienced. These traumas, such as alcohol-related accidents, homicides, and suicides, may be personally experienced or witnessed (Brave Heart and DeBruyn 1998; Halpern 2007; Steinman 2005). Native Americans not only are more likely to have violent or traumatic experiences that include serious injuries or threats of injuries but they also are more likely to witness such events. Compared to other ethnic groups, they experience the highest rate of violent victimization, and native teens are more likely than their non-native peers to be the victims of violent crime and assault (Sarche and Spicer 2008).

Increased stress from social and economic changes during the process of acculturation may have contributed to the high mortality rates from social pathologies, as has been suggested by studies among the Papago (Hackenberg and Gallagher 1972; Stull 1972, 1977) and Navajo (Broudy and May 1983). Not all researchers agree, however, and some authors have suggested that social pathologies are due to internal cultural dysfunctions rather than acculturation and social disruption (Levy and Kunitz 1971). There is little question that Native Americans have experienced extensive trauma since first contact with Europeans. Old World diseases and competition against the European newcomers for resources and land led to open conflict, colonization, and subjugation. U.S. government policies usurped Indian lands, forced Indians onto reservations and Indian children into boarding schools, and impoverished the people economically and culturally. Recent evidence suggests that stress experienced *in utero*, during the postnatal growth period and even in previous generations may have a real and transmissible effect on individual health and well-being (Benysheck 2001; Dabelea et al. 2000; Jasienska 2010; Kuzawa and Sweet 2009; Struthers and Lowe 2003).

Research suggests that diabetes, obesity, and depression are related (Anderson et al. 2000; Simon et al. 2006). Stress can impair glucose tolerance, and stress and other forms of psychological distress have a detrimental impact on glycemic control in diabetics. The stress hormone cortisol is antagonistic to insulin, contributing to high blood-sugar by encouraging gluconeogenesis in the liver (Plat et al. 1996). Diabetic patients are more likely than nondiabetics to suffer from clinical depression and are more than twice as likely to demonstrate significantly higher levels of depressive and other symptoms of psychological stress (Black 2002). Prevalence of depression among diabetics has been found to be highest among Native Americans, at 27.8% (Li et al. 2008). As a predictor, depression is somewhat problematic in that

it may be a cause as well as a result of an illness such as diabetes. Studies do indeed suggest that the relationship is bidirectional (Pan et al. 2010). In addition to the effects of stress on glucose tolerance, lipolysis may be mobilized in fat cells during times of stress, increasing cholesterol levels and contributing to the metabolic syndrome (Lönnqvist et al. 1992). Studies have also suggested that metabolic syndrome is a predictor of depression (Koponen et al. 2008).

Some researchers point to the public health system as responsible for the current health situation of Native Americans. Access to proper health care services is critical for controlling diabetes, preventing diabetic complications, and reducing mortality attributable to diabetes (Labarthe 1998). Kunitz (2004) cites the failure of the public health infrastructure to adequately address the rise of chronic disease conditions over the last few decades. Funding for Indian health has been inadequate and less per person than for the general U.S. public. Also, the health care system for Native Americans has failed to integrate into preventive services research findings that have identified risk factors associated with the most common causes of mortality. The inconsistent manner in which policies have been developed and implemented over the years has led to a schizophrenic and ineffective Indian policy that fails to inspire Native American confidence and is a primary feature of structural violence. The failure of public health services provided to Native Americans is no doubt a consequence of structural violence perpetrated against them by the institutions of the dominant society.

## **Chapter 5 References Cited**

Abourezk, J., Metcalf, L., Hatfield, M., Yates, S.R., Steiger, S., Young, D., Borbridge, J., Bruce, L.R., Deer, A., Dial, A., and Whitecrow, J. 1977. *American Indian Policy Review Commission Final Report, Submitted to Congress May 17, 1977*. American Indian Policy Review Commission. Washington, D.C.: U.S. Government Printing Office.

Adams, David W. 1995. *Education for Extinction: American Indians and the Boarding School Experience, 1875-1928*. Lawrence, Kansas: University Press of Kansas.



- Anderson, R.N. 2002. "Deaths: Leading Causes for 2000," in *National Vital Statistics Reports*, 50(16). Hyattsville, Maryland: National Center for Health Statistics.
- Anderson R.J., Lustman P.J., Clouse R.E., de Groot, M., and Freedland, K.E. 2000. Prevalence of Depression in Adults with Diabetes: A Systematic Review. *Diabetes* 49(Suppl 1):A64.
- Baker, J.P. and Katz, S.L. 2004. Childhood Vaccine Development: An Overview. *Pediatric Research* 55(2):347-356.
- Barker, D.J.P. 1994. *Mothers, Babies, and Disease in Later Life*. London: BMJ Publishing.
- Barnes, P.M., Adams, P.F., and Powell-Griner, E. 2005. *Health Characteristics of the American Indian and Alaska Native Adult Population: United States, 1999 – 2003. Advance Data from Vital and Health Statistics, No. 356*. Hyattsville, MD: National Center for Health Statistics.
- Barness, L.A., Opitz, J.M., and Gilbert-Barness, E. 2007. Obesity: Genetic, Molecular, and Environmental Aspects. *American Journal of Medical Genetics Part A* 143A:3016-3034.
- Barrett, J.R. 2011. A Different Diabetes: Arsenic Plus High-Fat Diet Yields an Unusual Diabetes Phenotype in Mice. *Environmental Health Perspectives* 119(8):A354.
- Beers, M.H., ed. 2005. "The Merck Manual of Medical Information," Second Home Edition, Online Version. Whitehouse Station, NJ: Merck Research Laboratories. May 16, 2005.  
<http://www.merck.com/mmhe/sec09/ch129/ch129c.html?qt=diarrhea&alt=sh#sec09-ch129-ch129c-712>
- Beers, M.H. and Berkow, R., eds. 1999. *The Merck Manual of Diagnosis and Therapy*. Whitehouse Station, NJ: Merck Research Laboratories.
- Benysheck, D.C. 2001. *The Political Ecology of Diabetes Among the Havasupai Indians of Northern Arizona*. PhD Dissertation. Arizona State University.
- Bergman, A.B., Grossman, D.C., Erdrich, A.M., Todd, J.G., and Forquera, R. 1999. A Political History of the Indian Health Service. *The Milbank Quarterly* 77:571-604.
- Berry, Mary F., Reynoso, C., Bracer, J.C., Edley, Jr., Kirsanow, P.N., Meeks, E.M., Redenbaugh, R.G., and Thernstrom, A. 2004. *Broken Promises: Evaluating the Native American Health Care System*. U.S. Commission on Civil Rights. Washington, D.C.: U.S. Government Printing Office.
- Berzok, L.M. 2005. *Food in American History: American Indian Food*. Westport, CT: Greenwood Publishing Group, Inc.
- Black, S.A. 2002. Diabetes, Diversity, and Disparity: What Do We Do with the Evidence? *American Journal of Public Health* 92(4):543-548.
- Bleich, S.N., Cutler, D., Murray, C., and Adams, A. 2008. Why is the Developed World Obese? *Annual Review of Public Health* 29:273-295.
- Bloomgarden, Z.T. 1998. Insulin Resistance: Current Concepts. *Clinical Therapeutics* 20(2):216-231.
- Brave Heart, M. and DeBruyn, L.M. 1998. The American Indian Holocaust: Healing Historical Unresolved Grief. *American Indian and Alaska Native Mental Health Research* 8:60-82.

- Broudy, D.W. and May P.A. 1983. Demographic and Epidemiologic Transition among the Navajo Indians. *Social Biology* 30(1):1-16.
- Bureau of the Census. 1924. *Mortality Statistics 1921*. Department of Commerce. Washington, DC: U.S. Government Printing Office.
- . 1927. *Mortality Statistics 1925*. Department of Commerce. Washington, DC: U.S. Government Printing Office.
- . 1932. *Mortality Statistics 1929*. Department of Commerce. Washington, DC: U.S. Government Printing Office.
- . 1937. *Mortality Statistics 1935*. Department of Commerce. Washington, DC: U.S. Government Printing Office.
- . 1943. *Vital Statistics of the United States 1940*. Department of Commerce. Washington, DC: U.S. Government Printing Office.
- Caballero, A.E. 2005. "Diabetes in Minorities in the United States", in *Joslin's Diabetes Mellitus*, 14<sup>th</sup> Edition. Edited by C. Kahn, G. Weir, G. King, A. Jacobson, A. Moses, and R. Smith, pp. 505-524. Boston: Joslin Diabetes Center.
- Campbell, G.R. 1989. The Changing Dimension of Native American Health: A Critical Understanding of Contemporary Native American Health Issues. *American Indian Culture and Research Journal* 13(3-4):1-20.
- . 1994. "Indian Health Service," in *Native America in the Twentieth Century: An Encyclopedia*. Edited by M. Davis, pp. 256-261. New York: Garland Publishing.
- Carter, M. 1916. Smallpox on an Indian Reservation. *The American Journal of Nursing* 17(2): 112-117.
- Carter, S.B. and Sutch, R. 2006. "American Indians," in *Historical Statistics of the United States, Millennial Edition*. Edited by S. Carter, S. Gartner, M. Haines, A. Olmstead, R. Sutch, and G. Wright, pp. 715-725. New York: Cambridge University Press.
- Castillo, E.D. 1978. *The Impact of Euro-American Exploration and Settlement. Handbook of North American Indians, Volume 3, California*. Edited by R. Heizer and W. Sturtevant, pp. 99-127. Washington, D.C.: Smithsonian Institution.
- Castor, Mei L., Smyser, Michael S., Taulii, Maile M., Park, Alice N., Lawson, Shelley A., and Forquera, Ralph A. 2006. A Nationwide Population-Based Study Identifying Health Disparities Between American Indians/Alaska Natives and the General Populations Living in Select Urban Counties. *American Journal of Public Health* 96:1478-1484.
- CDC (Centers for Disease Control and Prevention) 2003a. Health Status of American Indians Compared with Other Racial/Ethnic Minority Populations - Selected States, 2001 - 2002. *Morbidity and Mortality Weekly Report* 52(47): 1148-1152.

- . 2003b. Surveillance for Health Behaviors of American Indians and Alaska Natives, Findings from the Behavioral Risk Factor Surveillance System, 1997 - 2000. *Morbidity and Mortality Weekly Report* 52(SS-7).
- . 2004. Disparities in Premature Deaths from Heart Disease - 50 States and the District of Columbia, 2001. *Morbidity and Mortality Weekly Report* 53(6): 121-125.
- . 2012. Infant Mortality Statistics from the 2008 Period Linked Birth/Infant Death Data Set. *National Vital Statistics Reports* 60(5).
- Chase, Lillian A. 1937. The Trend of Diabetes in Saskatchewan, 1905 to 1934. *The Canadian Medical Association Journal* April: 366-369.
- Child, B.J. 1998. *Boarding School Seasons: American Indian Families, 1900-1940*. Lincoln, NE: University of Nebraska Press.
- Clifton, James, A. 1998. *The Prairie People: Continuity and Change in Potawatomi Indian Culture, 1665 - 1965*. Iowa City: University of Iowa Press,
- Codru, N., Schymura, M.J., Negoita, S. Rej, R., Carpenter, D.O. 2007. Diabetes in Relation to Serum Levels of Polychlorinated Biphenyls and Chlorinated Pesticides in Adult Native Americans. *Environmental Health Perspectives* 115(10):1442-1447.
- Collinson, John. 1936. Death Rates. *Annals of the American Academy of Political and Social Science, The American People: Studies in Population* 188: 84-93.
- Comstock, G. 1994. The International Tuberculosis Campaign: A Pioneering Venture in Mass Vaccination and Research. *Clinical Infectious Diseases* 19 (3):528-540.
- Connelley, W.E. 1918. The Prairie Band of Pottawatomie Indians. *Collections of the Kansas State Historical Society 1915-1918* 14:488-570.
- Conti, K.M. 2006. Diabetes Prevention in Indian Country: Developing Nutrition Models to Tell the Story of Food-System. *Journal of Transcultural Nursing* 17(3):234-245.
- Cordain, L., Eaton, S.B., Sebastian, A., Mann, N., Lindeberg, S., Watkins, B.A., O'Keefe, J.H., and Brand-Miller, J. 2005. Origins and Evolution of the Western Diet: Health Implications for the 21st century. *American Journal of Clinical Nutrition* 81:341-54.
- Dabelea, D., Hanson, R.L., Lindsay, R.S., Pettitt, D.J., Imperatore, G., Gabir, M.M., Roumain, J., Bennett, P.H., and Knowler, W.C. 2000. Intrauterine Exposure to Diabetes Conveys Risks for Type 2 Diabetes and Obesity: A Study of Discordant Sibships. *Diabetes* 49: 2008-2211.
- Dauer, C.C. 1943. Reported Whooping Cough Morbidity and Mortality in the United States. *Public Health Reports (1896-1970)* 58(17):661-676.
- de Cora, L. 2001. The Diabetic Plague in Indian Country: Legacy of Displacement. *Wicazo Sa Review, Native Health in the 21<sup>st</sup> Century* 16(1): 9-15.

- Deloria, Vine 1985. "The Evolution of Indian Policy Making," in *American Indian Policy in the Twentieth Century*. Edited by Vine Deloria, pp. 239-256. Norman: University of Oklahoma Press.
- DiBaise, J.K., Husen Zhang, H., Crowell, M.D., Krajmalnik-Brown, R., Decker, G.A., and Rittmann, B.E. 2008. Gut Microbiota and Its Possible Relationship with Obesity. *Mayo Clinic Proceedings* 83(4):460-469.
- Drewnowski, A. and Specter, S.E. 2004. Poverty and Obesity: The Role of Energy Density and Energy Costs. *American Journal of Clinical Nutrition* 79:6-16.
- Eaton, S.B., Shostak, M. and Konner, M. 1988. *The Paleolithic Prescription: A Program of Diet & Exercise and a Design for Living*. New York: HarperCollins.
- Enriori, P.J., Evans, A.E., Sinnayah, P., and Cowley, M.A. 2006. Leptin Resistance and Obesity. *Obesity* 14(Suppl.):254S-258S.
- Espey, D., Paisano, R. and Cobb, N. 2005 Regional Patterns and Trends in Cancer Mortality among American Indian and Alaska Natives, 1990–2001. *Cancer* 103:1045-1053.
- Flatz, G. 1987. "Genetics of Lactose Digestion in Humans," in *Advances in Human Genetics*. Edited by H. Harris and K. Hirschhorn, pp. 1-77. New York: Plenum Press.
- Flint, A.J. and Novotny, T.E. 1997. Poverty Status and Cigarette Smoking Prevalence and Cessation in the United States, 1983-1993: The Independent Risk of Being Poor. *Tobacco Control* 6:14-18.
- Ford, P.B. and Dzewaltowski, D.A. 2008. Disparities in Obesity Prevalence due to Variation in the Retail Food Environment: Three Testable Hypotheses. *Nutrition Reviews* 66(4):216–228.
- Freeman, W.L., Hosey, G.M., Diehr, P. Gohdes, D. 1989. Diabetes in American Indians of Washington, Oregon, and Idaho. *Diabetes Care* 12:282-288.
- Gluckman, P.D., Hanson, M.A., Cooper, C., and Thornburg, K.L. 2008. Effect of In Utero and Early-Life Conditions on Adult Health and Disease. *The New England Journal of Medicine* 359(1): 61-73.
- Gluckman, P.D., Hanson, M.A., and Spencer, H.G. 2005. Predictive Adaptive Responses and Human Evolution. *Trends in Ecology and Evolution* 20(10): 527-533.
- Gohdes, D. 1995. "Diabetes in North American Indians and Alaska Natives," in *Diabetes in America*. National Diabetes Data Group, NIH Publication No. 95-1468, 2nd ed., pp. 683-701. Bethesda, MD: National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health.
- Grarup, N., Sparsø, T., and Hansen, T. 2010. Physiologic Characterization of Type 2 Diabetes–Related Loci. *Current Diabetes Reports* 10:485-497.
- Guyon, S. 1973. The Challenge to the Indian Health Service. *Health Services Reports* 88(8):687-691.
- Hackenberg, R.A. 1966. An Anthropological Study of Demographic Transition: The Papago Information System. *The Milbank Memorial Fund Quarterly* 44(4) Part 1:470-493.

Hackenberg, R.A., and Gallagher, Mary M. 1972. The Costs of Cultural Change: Accidental Injury and Modernization among the Papago Indians. *Human Organization* 31:211-226.

Hall, T.R., Hickey, M.E., and Young, T.B. 1992. Evidence for Recent Increases in Obesity and Non-Insulin-Dependent Diabetes Mellitus in a Navajo Community. *American Journal of Human Biology* 4(4):547-553.

Halpern, P. 2007. *Obesity and American Indians/Alaska Natives*. Report Prepared for the U.S. Department of Health and Human Services, Office of the Assistant Secretary for Planning and Evaluation.

Hanson, R.L., Bogardus, C., Duggan, D., Kobes, S., Knowlton, M., Infante, A.M., Marovich, L., Benitez, D., Baier, L.J., and Knowler, W.C. 2007. A Search for Variants Associated With Young-Onset Type 2 Diabetes in American Indians in a 100K Genotyping Array. *Diabetes* 56:3045-3052.

Heid, I.M. et al. 2010. Meta-analysis identifies 13 new loci associated with waist-hip ratio and reveals sexual dimorphism in the genetic basis of fat distribution. *Nature Genetics* 42(11):949-960.

Helmrich, S.P., Ragland, D.R., Leung, R.W., Paffenbarger Jr., R.S. 1991. Physical Activity and Reduced Occurrence of Non-Insulin-Dependent Diabetes Mellitus. *The New England Journal of Medicine* 325(3): 147-152.

Hill Jr., C.A. 1970. Measures of Longevity of American Indians. *Public Health Reports (1896-1970)* 85(3):233-239.

Hill Jr., C.A. and Spector, M.I. 1971. Natality and Mortality of American Indians Compared with U.S. Whites and Nonwhites. *HSMHA Health Reports* 86(3):229-246.

IHS (Indian Health Service) 1974. *Indian Health Trends and Services, 1974 Edition*. Washington, DC: U.S. Department of Health, Education, and Welfare.

—. 1979. *Selected Vital Statistics for Indian Health Service Areas and Service Units, 1972 – 1977*. Rockville, MD: U.S. Department of Health, Education, and Welfare.

—. 1998. *Trends in Indian Health, 1997*. Rockville, MD: U.S. Department of Health and Human Services.

—. 2001. IHS Report to Congress: Obesity Prevention and Control for American Indians and Alaska Natives. <http://www.ihs.gov/hpdp/Documents/ObesityPreventionReport.pdf>. May 1<sup>st</sup> 2011.

—. 2004. *Trends in Indian Health, 2000-2001*. Rockville, Md.: U.S. Department of Health and Human Services.

—. 2005. *The First 50 Years of the Indian Health Service: Caring and Curing*. Rockville, Md.: U.S. Department of Health and Human Services.

—. 2009. *Trends in Indian Health, 2002-2003*. Rockville, Md.: U.S. Department of Health and Human Services.

Ivy, J.L. 1997. Role of Exercise Training in the Prevention and Treatment of Insulin Resistance and Non-Insulin-Dependent Diabetes Mellitus. *Sports Medicine* 24(5): 321-336.

Jackson, H.M., Bible, A., Church, F., Metcalf, L., Johnston, J.B., Abourezk, J., Haskell, F.K., Nelson, G., Metzenbaum, H.M., Fannin, P.L., Hansen, C.P., Hatfield, M.O., Buckley, J.L., McClure, J.A., Bartlett, D.F. 1974. *Indian Health Care Improvement Act: Report of the Committee on Interior and Insular Affairs, United States Senate together with Additional Views to Accompany S.2938*. Report No. 93-1283. Washington, D.C.: U.S. Government Printing Office.

Jasienska, G. 2010. "Why Women Differ in Ovarian Function: Genetic Polymorphism, Developmental Conditions, and Adult Lifestyle" in *Human Evolutionary Biology*. Edited by M. Muehlenbein, pp. 322-337. New York: Cambridge University Press.

Johnson, A.E. and Taylor, A.K. 1991. *Prevalence of Chronic Diseases: A Summary of Data from the Survey of American Indians and Alaska Natives*. Pub. No. 91-0031. Rockville, MD: Agency for Health Care Policy and Research.

Kane, Robert L., and Kane, Rosalie A. 1972. *Federal Health Care (With Reservations!)*. New York: Springer Publishing Company.

Keller, J.A. 2002. *Empty Beds: Indian Student Health at Sherman Institute, 1902 - 1922*. East Lansing: Michigan State University Press.

Klimentidis, Y.C., Miller, G.F., and Shriver, M.D. 2009. The Relationship Between European Genetic Admixture and Body Composition among Hispanics and Native Americans. *American Journal of Human Biology*, 21:377-382.

Knowler, W.C., Bennett, P.H., Hamman, R.F., Miller, M. 1978. Diabetes Incidence and Prevalence in Pima Indians: A 19-Fold Greater Incidence than in Rochester, Minnesota. *American Journal of Epidemiology* 108:497-505.

Knowler, W.C., McCance, D.R., Nagi, D.K., and Pettitt, D.J. 1993. "Epidemiological Studies of the Causes of Non-Insulin Dependent Diabetes Mellitus," in *Causes of Diabetes: Genetic and Environment Factors*. Edited by R. Leslie, pp.187-218. Chinchester, England: Wiley & Sons.

Knowler, W.C., Pettitt, D.J., Bennett, P.H., and Williams, R.C. 1983. Diabetes Miletus in the Pima Indians: Genetic and Evolutionary Considerations. *American Journal of Physical Anthropology* 62(1):107-114.

Knowler, W.C., Pettitt, D.J., Saad, M.F., and Bennett, P.H. 1990. Diabetes Mellitus in the Pima Indians: Incidence, Risk Factors and Pathogenesis. *Diabetes Metabolism Reviews* 6:1-27.

Knowler, W.C., Pettit, D.J., Savage, P.J., and Bennett, P.H. 1981. Diabetes Incidence in Pima Indians: Contributions of Obesity and Parental Diabetes. *American Journal of Epidemiology* 113(3):144-155.

Knowler, W.C., Saad, M.F., Pettitt, D.J., Nelson, R.G., Bennett, P.H. 1993. Determinants of Diabetes Mellitus in the Pima Indians. *Diabetes Care* 16:216-227.

Koponen, H., Jokelainen, J., Keinanen-Kiukaanniemi, S., Kumpusalo, E., and Vanhala, M. 2008. Metabolic Syndrome Predisposes to Depressive Symptoms: A Population-Based 7-Year Follow-Up Study. *Journal of Clinical Psychology* 23:e1-e5.

- Kuhnlein, H.V. and Receveur, O. 1996. Dietary Change and Traditional Food Systems of Indigenous Peoples. *Annual Review of Nutrition* 16:417-442.
- Kunitz, S.J. 2004. "The Evolution of Disease and the Devolution of Health Care for American Indians," in *The Changing Face of Disease: Implications for Society*. Edited by N. Mascie-Taylor, J. Peters, and S. McGarvey, pp. 153-169. Society for the Study of Human Biology Series: 43. Boca Raton, FL: Routledge.
- Kuzawa, C.W. and Sweet, E. 2009. Epigenetics and the Embodiment of Race: Developmental Origins of US Racial Disparities in Cardiovascular Health. *American Journal of Human Biology* 21:2-15.
- Kuzawa, C.W. and Thayer, Z.M. 2011. Timescales of Human Adaptation: The Role of Epigenetic Processes. *Epigenomics* 3(2):221-234.
- Labarthe, D.R. 1998. *Epidemiology and Prevention of Cardiovascular Disease: A Global Challenge*. Gaithersburg, Md: Aspen Publications.
- Lang, Gretchen C. 1985. Diabetics and Health Care in a Sioux Community. *Human Organization* 44:251- 260.
- Lappe, M. 1994. *Evolutionary Medicine: Rethinking the Origins of Disease*. San Francisco: Sierra Club Books.
- Lee, Elisa T., Cowan, Linda D., Welty, T.K., Sievers, M., Howard, William J., Oopik, A., Wang, W., Yen, J., Devereux, R.B., Rhoades, E.R., Fabsitz, R.R., Go, O., and Howard, B.V. 1998. All-Cause Mortality and Cardiovascular Disease Mortality in Three American Indian Populations, Aged 45-74 Years, 1984-1988: The Strong Heart Study. *American Journal of Epidemiology* 147(11):995-1008.
- Levison, C.H., Hastings, D.W., and Harrison, J.N. 1981. Epidemiological Transition in a Frontier Town - Manti, Utah: 1849 - 1977. *American Journal of Physical Anthropology* 56:83-93.
- Levy, J.E. and Kunitz, S.J. 1971. Indian Reservations, Anomie, and Social Pathologies. *Southwestern Journal of Anthropology* 27(2):97-128.
- Li, C., Ford, E.S., Strine, T.W., and Mokdad, A.H. 2008. Prevalence of Depression among U.S. Adults with Diabetes: Findings from the 2006 Behavioral Risk Factor Surveillance System. *Diabetes Care* 31(1):105-107.
- Löhn, C. 1995 *Prevention of Non-Insulin-Dependent Diabetes Mellitus (NIDDM) among the Southern Cheyenne: An Analysis of its Prevalence, Risk Factors and Initial Treatment among Full-Blood Indians*. Ph.D. dissertation, Department of Anthropology, University of Kansas.
- Lönnqvist, F., Wennlund, A., Wahrenberg, H., and Arner, P. 1992. Effects of Mental Stress on Lipolysis in Humans. *Metabolism* 41(6):622-630.
- Ludwig, J., Sanbonmatsu, L., Gennetian, L., Adam, E., Duncan, G.J., Katz, L.F., Kessler, R.C., Kling, J.R., Lindau, S.T., Whitaker, R.C., and McDade, T.W. 2011. Neighborhoods, Obesity, and Diabetes — A Randomized Social Experiment. *New England Journal of Medicine* 365:1509-1519.
- Lux, M.K. 2001. *Medicine that Walks: Disease, Medicine, and Canadian Plains Native People, 1880 – 1940*. Toronto: University of Toronto Press.

Lynch, J.W., Kaplan, G.A., and Shema, S.J. 1997. Cumulative Impact of Sustained Economic Hardship on Physical, Cognitive, Psychological, and Social Functioning. *New England Journal of Medicine* 337:1889–1895.

Ma, Lijun, Hanson, R.L., Que, L.N., Guo, Y., Kobes, S., Bogardus, C., and Baier, L.J. 2008. PCLO Variants Are Nominally Associated With Early-Onset Type 2 Diabetes and Insulin Resistance in Pima Indians. *Diabetes* 57 (2008):3156-3160.

Manson, S.M. and Altschul, D.B. 2004. *Cultural Diversity Series: Meeting the Mental Health Needs of American Indians and Alaska Natives*. National Technical Assistance Center for State Mental Health Planning (NTAC), and the National Association of State Mental Health Program Directors (NASMHPD). Rockville, MD: U.S. Department of Health and Human Services.

Marrett, L.D. and Chaudhry, M. 2003. Cancer Incidence and Mortality in Ontario First Nations, 1968 - 1991. *Cancer Causes and Control* 14(3):259-268.

Massachusetts General Hospital (2010). Genetics of obesity and fat distribution: Apple and pear shapes partly due to genes. ScienceDaily.  
<http://www.sciencedaily.com/releases/2010/10/101010133620.htm>. November 15<sup>th</sup>, 2011.

Mayer-Davis, E.J., D'Agostino, R.Jr., Karter, A.J., Haffner, S.M., Rewers, M.J., Saad, M., and Bergman, R.N. 1998. Intensity and Amount of Physical Activity in Relation to Insulin Sensitivity: The Insulin Resistance Atherosclerosis Study. *JAMA* 279(9):669-674.

McMillen, C.W. 2008. The Red Man and the White Plague: Rethinking Race, Tuberculosis, and American Indians, ca. 1890-1950. *Bulletin of the History of Medicine* 82(3):608-645.

Meriam, L., Brown, R.A., Roe Cloud, H., Dale, E.E., Duke, E., Edwards, H.R., McKenzie, F.A., Mark, M.L., Ryan Jr., W.C., and Spillman, W.J. 1928. *The Problem of Indian Administration*. Report of a Survey at the Request of Honorable Hubert Work, Secretary of the Interior, and Submitted to Him, February 21, 1928. Washington, D.C.: Institute for Government Research.

Michel, K.L. 2004. *The New Focus on Native American Cooking*. The Washington Post, September 22, 2004, F01.

Mihesuah, Devon A. 2003. Decolonizing Our Diets by Recovering Our Ancestors' Gardens. *American Indian Quarterly* 27(3/4):807-839.

Morell, S.F. and Enig, M. 2001. Guts and Grease: The Diet of Native Americans. *Wise Traditions in Food, Farming, and the Healing Arts* 2(1):1-15.

Mouratoff, G.J., Carroll, N.V., and Scott E.M. 1967. Diabetes Mellitus in Eskimos. *JAMA* 199:107-112.

Mouratoff, G.J. and Scott, E.M. 1973. Diabetes Mellitus in Eskimos after a Decade. *JAMA* 266:1345-1346.

Mulligan, C.J., Robin, R.W., Osier, M.V., Sambughin, N., Goldfarb, L.G., Kittles, R.A., Hesselbrock, D., Godlman, D., and Long, J.C. 2003. Allelic Variation at Alcohol Metabolism Genes (ADH1B,



ADH1C, ALDH2) and Alcohol Dependence in an American Indian Population. *Human Genetics* 113:325-336.

NCHS (National Center for Health Statistics). 1963. *Vital Statistics of the United States, 1960*. U.S. Public Health Service. Washington, DC: U.S. Department of Health, Education, and Welfare.

—. 1964. *Vital Statistics of the United States, 1961*. U.S. Public Health Service. Washington, DC: U.S. Department of Health, Education, and Welfare.

—. 1974. *Vital Statistics of the United States, 1971*. U.S. Public Health Service. Washington, DC: U.S. Department of Health, Education, and Welfare.

—. 1977. *Vital Statistics of the United States, 1975*. U.S. Public Health Service. Washington, DC: U.S. Department of Health, Education, and Welfare.

—. 2005. *Health, United States 2005: With Chartbook on Trends in the Health of Americans*. Hyattsville, MD: U.S. Department of Health and Human Services.

NIAAA (National Institute on Alcohol Abuse and Alcoholism). 2002. Alcohol and Minorities: An Update. Alcohol Alerts, No. 55. National Institutes of Health.

<http://pubs.niaaa.nih.gov/publications/aa55.htm> November 1<sup>st</sup>, 2001.

NOVS (National Office of Vital Statistics). 1947. *Vital Statistics of the United States 1945*. U.S. Public Health Service. Washington, DC: U.S. Government Printing Office.

—. 1953. *Vital Statistics of the United States 1950*. U.S. Public Health Service, U.S. Department of Health, Education, and Welfare. Washington, DC: U.S. Government Printing Office.

—. 1957. *Vital Statistics of the United States 1955*. U.S. Public Health Service, U.S. Department of Health, Education, and Welfare. Washington, DC: U.S. Government Printing Office.

Newman, J.M., Marfin, A.A., Eggers, P.W., and Helgersen, S.D. 1990. End Stage Renal Disease among Native Americans, 1983 – 1986. *American Journal of Public Health* 80:318-319.

North, K.E., MacCluer, J.W., Williams, J.T., Welty, T.K., Best, L.G., Lee, E.T., Fabsitz, R.R., and Howard, B.V. 2003. Evidence for Distinct Genetic Effects on Obesity and Lipid-Related CVD Risk Factors in Diabetic Compared to Nondiabetic American Indians: the Strong Heart Family Study. *Diabetes Metabolism Research and Reviews* 19:140-147.

Omran, A.R. 1971 The Epidemiologic Transition; A Theory of the Epidemiology of Population Change. *The Milbank Memorial Fund Quarterly* 49(4):509-538.

—. 1977. A Century of Epidemiologic Transition in the United States. *Preventive Medicine* 6(1):30-51.

Paltoo, D.N. and Chu, K.C. Patterns in Cancer Incidence among American Indians/Alaska Natives, United States, 1992-1999. *Public Health Reports (1974-)* 119(4):443-451.

Pan, A., Lucas, M., Sun, Q., van Dam, R.M., Franco, O.H., Manson, J.E., Willett, W.C., Ascherio, A., and Hu, F. 2010. Bidirectional Association between Depression and Type 2 Diabetes Mellitus in Women. *Archives of Internal Medicine* 170(21):1884-1891.

- Parra, E.J., Hoggart, C.J., Bonilla, C., Dios, S., Norris, J.M., Marshall, J.A., Hamman, R.F., Ferrell, R.E., McKeigue, P.M., and Shriver M.D. 2004. Relation of Type 2 Diabetes to Individual Admixture and Candidate Gene Polymorphisms in the Hispanic American Population of San Luis Valley, Colorado. *Journal of Medical Genetics* 41:e116.
- Paul, D.S., Walton, F.S., Saunders, R.J., and Stýblo, M. 2011. Characterization of the Impaired Glucose Homeostasis Produced in C57BL/6 Mice by Chronic Exposure to Arsenic and High-Fat Diet. *Environmental Health Perspectives* 119(8):1104-1109.
- Perrott, George St. J. and West, M.D. 1957. Health Services for American Indians. *Public Health Reports (1896-1970)* 72(7):565-570.
- Plat, L., Byrne, M.M., Sturis, J., Polonsky, K.S., Mockel, J., and Van Cauter, E. 1996. Effects of Morning Cortisol Elevation on Insulin Secretion and Glucose Regulation in Humans. *American Journal of Physiology* 270(1):E36-42.
- Popkin, B.M. 1993. Nutrition Patterns and Transitions. *Population and Development Review* 19:138-157.
- PRC (Population Resource Center). 2006. Factsheet: American Indian and Alaska Native Heritage Month. Washington, D.C.: Population Resource Center.
- Prucha, Francis P. 1976. *American Indian Policy in Crisis: Christian Reformers and the Indian, 1865 – 1900*. Norman, OK: University of Oklahoma Press.
- . 1984a. American Indian Policy in the Twentieth Century. *The Western Historical Quarterly* 15(1):4-18.
- . 1984b. *The Great Father: The United States Government and the American Indians*, Vols. 1 and 2. Lincoln: University of Nebraska Press.
- Putney, Diane T. 1980. *Fighting the Scourge: American Indian Morbidity and Federal Policy, 1897 – 1928*. PhD Dissertation. Milwaukee, WI: Marquette University.
- Quiggins, P.A. 1990. *Non-Insulin-Dependent (Type II) Diabetes Mellitus in the Eastern Cherokee of Western North Carolina*. PhD Dissertation. The University of Tennessee.
- Ravussin, E. 1995. Metabolic Differences and the Development of Obesity. *Metabolism* 44(9) Suppl 3:12-14.
- Reed, T.E. 1985. Ethnic Differences in Alcohol Use, Abuse, and Sensitivity: A Review with Genetic Interpretation. *Social Biology* 32:195-209.
- Reeves, M.J., Remington, P.L., Nashold, R., and Pete, J. 1997. Chronic Disease Mortality among Wisconsin Native American Indians, 1984 – 1993. *Wisconsin Medical Journal* 96(2):27-32.
- Rhoades, Everett R. 2003. The Health Status of American Indian and Alaska Native Males. *American Journal of Public Health* 93:774-778.
- Richards, T.J., and Patterson, P.M. 2006. Native American Obesity: An Economic Model of the “Thrifty Gene” Theory. *American Journal of Agricultural Economics* 88:542-560.

- Ringhand, T.H., Snowdon, D.A., and Johnson, R.A. 1990. Trends in Rates for Mortality from All Causes among Indians in Minnesota, 1960-79. *Public Health Reports* 105(4):425-428.
- Rith-Najarian, S.J., Gohdes, D.M., Shields, R., Skipper, B., Moore, K.R., Tolbert, B., Raymer, T., and Acton, K.J. 2002. Regional Variation in Cardiovascular Disease Risk Factors among American Indians and Alaska Natives with Diabetes. *Diabetes Care* 25(2):279-283.
- Robbins, J.M., Vaccarino, V., Zhang, H., and Kasl, S. 2001. Socioeconomic Status and Type 2 Diabetes in African American and Non-Hispanic White Women and Men: Evidence from the Third National Health and Nutrition Examination Survey. *American Journal of Public Health* 91:76-83.
- Rosenberg, T. and Martel, S. 1998. Cancer Trends from 1972 – 1991 for Registered Indians Living on Manitoba Reserves. *International Journal of Circumpolar Health* 57(1) Supplement:391-398.
- Rowley, D.L. and MacDorman, M.F. 1994. “Neonatal and Postneonatal Mortality,” in *From Data to Action: CDC’s Public Health Surveillance for Women, Infants, and Children*. Atlanta, GA: Centers for Disease Control and Prevention.
- Saglid, U. Littauer, J., Jespersen, C.S., and Anderson, S. 1966. Epidemiological Studies in Greenland 1962 - 1964. I. Diabetes Mellitus in Eskimos. *Acta Medica Scandinavica* 179:29-39.
- Sarche, M. and Spicer, P. 2008. Poverty and Health Disparities for American Indian and Alaska Native Children: Current Knowledge and Future Prospects. *Annals of the New York Academy of Sciences* 1136:126-136.
- Schraer, C.D., Lanier, A.P., Boyko, E.J., Gohdes, D., and Murphy, N.J. 1988. Prevalence of Diabetes Mellitus in Alaskan Eskimos, Indians, and Aleuts. *Diabetes Care* 11:693-700.
- Schultz, L.O., Bennett, P.H., Ravussin, E., Kidd, J.R., Kidd, K.K., Esparza, J., and Valencia, M.A. 2006. Effects of Traditional and Western Environments on Prevalence of Type 2 Diabetes in Pima Indians in Mexico and the U.S. *Diabetes Care* 29(8):1866-1871.
- Scott, E.M. and Griffith, I.V. 1957. Diabetes Mellitus in Eskimos. *Metabolism* 6:320-325.
- Shaffer, E.A. 2005. Epidemiology and Risk Factors for Gallstone Disease: Has the Paradigm Changed in the 21st Century? *Current Gastroenterology Reports* 7(2):132-140.
- Sievers, M.L. 1966. Disease Patterns among Southwestern Indians. *Public Health Reports (1896-1970)* 81(12):1075-1083.
- Sievers, M.L. and Fisher, J.R. 1983. Cancer in North American Indians: Environment versus Heredity. *American Journal of Public Health* 73(5):485-487.
- Sievers M.L., Nelson R.G., and Bennett P.H. 1996. Sequential trends in overall and cause-specific mortality in diabetic and nondiabetic Pima Indians. *Diabetes Care* 19:107-111.
- Simon G.E., von Korff M., Saunders K., Miglioretti D.L., Crane P.K., van Belle G., and Kessler R.C. 2006. Association between Obesity and Psychiatric Disorders in the US Adult Population. *Archives of General Psychiatry* 63:824-830.

Singh, G.K., and Hoyert, D.L. 2000. Social Epidemiology of Chronic Liver Disease and Cirrhosis Mortality in the United States, 1935 – 1997: Trends and Differentials by Ethnicity, Socioeconomic Status, and Alcohol Consumption. *Human Biology* 72(5):801-820.

Smith-Morris, Carolyn M. 2004. Reducing Diabetes in Indian Country: Lessons from the Three Domains Influencing Pima Diabetes. *Human Organization* 63:34-46.

Snipp, C. Matthew 2006. "Life Expectancy at Birth for American Indians, by Sex: 1940–1993," in *Historical Statistics of the United States, Earliest Times to the Present: Millennial Edition*, Edited by S. Carter, S. Gartner, M. Haines, A. Olmstead, R. Sutch, and G. Wright, Table A539-541. New York: Cambridge University Press.

Speakman, J.R., Rance, K.A., and Johnstone, A.M. 2008. Polymorphisms of the FTO Gene are Associated with Variation in Energy Intake, but not Energy Expenditure. *Obesity* 16(8):1961-1965.

Steinman, E. 2005. Native Americans suffer from historical trauma, researcher says. The United Methodist Church News, July 27, 2005.  
[http://www.umc.org/site/c.gjJTJbMUluE/b.928147/k.CB36/Native\\_Americans\\_suffer\\_from\\_historical\\_trauma\\_researcher\\_says.htm](http://www.umc.org/site/c.gjJTJbMUluE/b.928147/k.CB36/Native_Americans_suffer_from_historical_trauma_researcher_says.htm). December 19<sup>th</sup> 2011.

Sterling, E.B. 1933. Maternal, Fetal, and Neonatal Mortality among 1,815 Hospitalized American Indians. *Public Health Reports (1896-1970)* 48(20):522-535.

Story, M., Strauss, K., Gilbert, T.J., & Broussard, B.A. 2000. "Nutritional Health and Diet-Related Conditions," in *American Indian Health*. Edited by E.R. Rhoades, pp. 201-220. Baltimore, MD: The Johns Hopkins University Press.

Struthers, R. and Lowe, J. 2003. Nursing in the Native American Culture and Historical Trauma. *Issues in Mental Health Nursing* 24:257-272.

Stull, D.D. 1972. Victims of Modernization: Accident Rates and Papago Indian Adjustment. *Human Organization* 31(2):227-240.

—. 1977. New Data on Accident Victims Rates among the Papago Indians: The Urban Case. *Human Organization* 36(4):395-398.

Swinburn, B.A., Nyomba, B.L., Saad, M.F., Zurlo, F., Raz, I., Knowler, W.C., Lillioja, S., Bogardus, C., and Ravussin, E. 1991. Insulin Resistance Associated with Lower Rates of Weight Gain in Pima Indians. *Journal of Clinical Investigation* 88:168-173.

Szathmary, E.J. 1990. "Diabetes in Amerindian Populations: The Dogrib Studies," in *Disease in Populations in Transition: Anthropological and Epidemiological Perspectives*. Edited by A. Swedlund and G. Armelagos, pp. 75-103. New York: Bergin and Garvey.

—. 1994a. "Factors that Influence the Onset of Diabetes in Dogrib Indians of the Canadian Northwest Territories," in *Diabetes as a Disease of Civilization: The Impact of Culture Change on Indigenous Peoples*. Edited by J. Joe and R. Young, pp. 229-268. New York: Mouton de Gruyter.

—. 1994b. Non-Insulin Dependent Diabetes Mellitus among Aboriginal North Americans. *Annual Review of Anthropology* 23:457-482.

- Thomas, K.T., Keller, C.S., and Holbert, K.E. 1997. Ethnic and Age Trends for Body Composition in Women Residing in the U.S. Southwest: I. Regional Fat. *Medicine and Science in Sport and Exercise* 29(1):82-89.
- Thorleifsson, G., et al. 2009. Genome-Wide Association Yields New Sequence Variants at Seven Loci that Associate with Measures of Obesity. *Nature Genetics* 41(1):18-24.
- Townsend, J.G. 1938. Disease and the Indian. *The Scientific Monthly* 47(6):479-495.
- Trafzer, C.E. 1997. *Death Stalks the Yakama: Epidemiological Transitions and Mortality on the Yakama Indian Reservation, 1888-1964*. East Lansing, MI: Michigan State University Press.
- Trovato, F. 1988. Mortality Differentials in Canada, 1951-1971: French, British, and Indians. *Culture, Medicine and Psychiatry* 12:459-477.
- Tyler, S. Lyman 1973. *A History of Indian Policy*. Washington, D.C.: Bureau of Indian Affairs, United States Department of the Interior, U.S. Government Printing Office.
- Tyroler, H.A. and Patrick, R. 1972 Epidemiologic Studies of Papago Indian Mortality. *Human Organization* 31:163-170.
- USPHS (U.S. Public Health Service). 1913. *The Prevalence of Contagious and Infectious Diseases among the Indians of the United States*. Senate Document No. 1038, 62<sup>nd</sup> Congress, 3<sup>rd</sup> session, serial 6365.
- Wallace, Helen M. 1972. The Health of American Indian Children: A Summary of the Literature. *Health Services Reports* 87(9):867-876.
- Walston, J., Silver, K., Bogardus, C., Knowler, W.C., Celi, F.S., Austin, S., Manning, B., Strosberg, A.D., Stern, M.P., Raben, N., Sorkin, J.D., Roth, J., and Shuldiner, A.R. 1995. Time onset of non-insulin-dependent diabetes mellitus and genetic variation in the  $\beta 3$ -adrenergic-receptor gene. *New England Journal of Medicine* 333:343-347.
- Wareharn, N.J. 2004. "Unraveling Gene-Environment Interactions in Type 2 Diabetes," in *The Changing Face of Disease: Implications for Society*. Edited by N. Mascie-Taylor, J. Peters, and S. McGarvey, pp. 130-138. Society for the Study of Human Biology Series: 43. Boca Raton, FL: Routledge.
- Wallace, Helen M. 1972. The Health of American Indian Children: A Summary of the Literature. *Health Services Reports* 87(9):867-876.
- Weiss, K.M. 1990. "Transitional Diabetes and Gallstones in Amerindian Peoples: Genes of Environment?" in *Disease in Populations in Transition: Anthropological and Epidemiological Perspectives*. Edited by A. Swedlund and G. Armelagos, pp. 105-123. New York: Bergin and Garvey.
- Weiss, K.M., Ferrell, R.E., and Hanis, C.L. 1984. A New World Syndrome of Metabolic Diseases with a Genetic and Evolutionary Basis. *Yearbook of Physical Anthropology* 27:153-178.
- Weiss, K.M., Ferrell, R.E., Hanis, C.L., and Styne, P.N. 1984. Genetics and Epidemiology of Gallbladder Disease in New World Native Peoples. *American Journal of Human Genetics* 36:1259 - 1278.

Weiss, K.M., Ulbrecht, J.S., Cavanagh, P.R., and Buchanan, A.V. 1989. Diabetes Mellitus in American Indians: Characteristics, Origins, and Preventive Health Care Implications. *Medical Anthropology* 11(3):283-304.

West, K.M. 1974. Diabetes in American Indians and Other Native Populations of the New World. *Diabetes* 23:841-855.

Westfall, D.N. and Rosenbloom, A.L. 1971. Diabetes Mellitus among the Florida Seminoles. *HSMHA Health Reports* 86(11):1037-1041.

Willer, C.J. et al. 2009. Six New Loci Associated with Body Mass Index Highlight a Neuronal Influence on Body Weight Regulation. *Nature Genetics* 41(1):25–34.

Wirsing, R.L., Logan, M.H., Micozzi, M.S., Nyamwaya, D.O., Pearce, T.O., Renshaw, D.C., and Schaefer, O. 1985. The Health of Traditional Societies and the Effects of Acculturation. *Current Anthropology* 26(3):303-322.

Wissler, Clark. 1936. The Effect of Civilization upon the Length of Life of the American Indian. *The Scientific Monthly* 43(1):5-13.

Wolsey, Darcy H., and Cheek, James, E. 1999. "Epidemiologic Patterns of Morbidity and Mortality," in *Primary Care of Native American Patients: Diagnosis, Therapy, and Epidemiology*. Edited by J.M. Galloway, B.W. Goldberg, and J.S. Alpert, pp. 7-16. Boston: Butterworth Heinemann.

Yach, D., Stuckler, D., and Brownwell, K.D. 2006. Epidemiologic and Economic Consequences of the Global Epidemics of Obesity and Diabetes. *Nature Medicine* 12(1):62-66.

Young, T.K. 1988. Are Subarctic Indians Undergoing the Epidemiologic Transition? *Social Science and Medicine*. 26(6):659-671.

—. 1994. *The Health of Native Americans: Toward a Biocultural Epidemiology*. Oxford: Oxford University Press.

—. 1996. "Recent Health Trends in the Native American Population," *Changing Numbers, Changing Needs: American Indian Demography and Public Health*. Edited by G. Sandefur and B. Cohen, pp. 53-75. Washington, D.C.: National Academy Press.

\*Also published in *Population Research and Policy Review* 1997. 16:146-147.

Young, T. K., McIntyre, L. L., Dooley, J. and Rodrituez, J. 1985. Epidemiologic Features of Diabetes Mellitus among Indians in Northwestern Ontario and Northeastern Manitoba. *Canadian Medical Association* 132: 793–797.

Young, T.K., Schraer, C.D., Shubnikoff, E.V., Szathmary, E.J., and Nikitin, Y.P. 1992. Prevalence of Diagnosed Diabetes in Circumpolar Indigenous Populations. *International Journal of Epidemiology* 21:730-736.

Zurlo, F., Lillioja, S., Esposito-Del Puente, A., Nyomba B.L., Raz, I., Saad, M.F., Swinburn, B.A., Knowler, W.C., Bogardus, C., Ravussin E. 1990. Low ratio of fat to carbohydrate oxidate as predictor of weight gain: study of 24-h RQ. *American Journal of Physiology* 259:E650-E657.

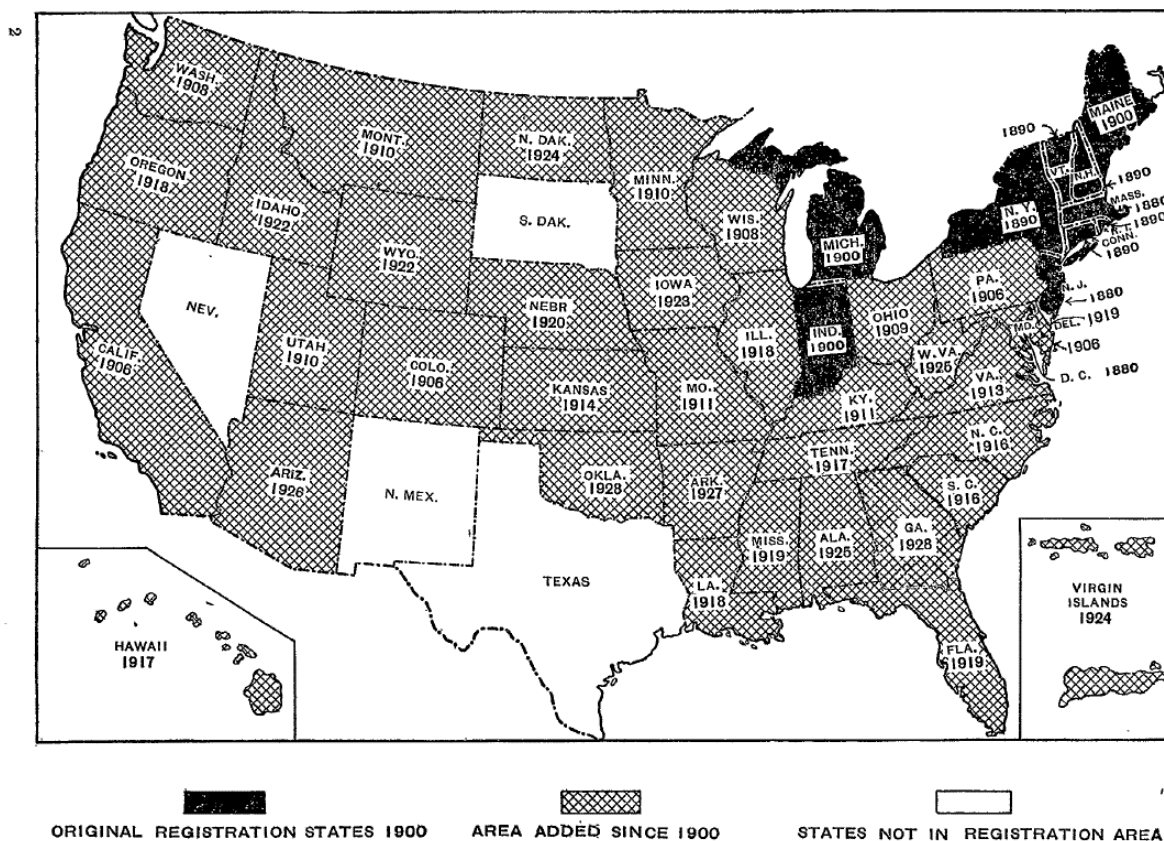
## **Chapter 6: Health and Disease among Prairie Band Potawatomi in the early 20th Century. Part I. The Reports of the Potawatomi Agency**

Administrative reports of the Potawatomi Agency provide earliest information on the health conditions of the tribe after relocation to Kansas. The Potawatomi Agency in Mayetta, Kansas, was established in 1851 for the Potawatomi and, until 1855, for the Konza (Kaw) Indians in Kansas. By 1871 the Prairie Band was the only Potawatomi group remaining in the state. In 1874, the Kickapoo Agency was consolidated into the Potawatomi Agency, making it the only Indian agency in Kansas, so it was referred to as the Kansas Agency. The Chippewa and Munsee, who for some years had not had an agency, were assigned to the Potawatomi Agency in 1876. In 1882, the Potawatomi Agency and the Great Nemaha Agency for the Sac and Fox of Missouri and the Iowa of Nebraska were merged, forming the Potawatomi and Great Nemaha Agency. In 1903, the agency was reconfigured into two separate agencies: the Potawatomi Agency for the Potawatomi; and the Kickapoo Agency for the Kickapoo, Sac and Fox, and Iowa. They were again combined in 1919 as the Kickapoo Agency, which, in 1921, was renamed the Potawatomi Agency. From 1927 until 1935, the agency was consolidated with Haskell Institute in Lawrence, Kansas, after which it once again became independent. The agency was renamed the Potawatomi Area Field Office in 1950, and in 1964 the name was changed again to the Horton Agency (Hill, 1981; NARA 2009).

### **Methods**

#### **Data**

Kansas became a Registration State for the Mortality Statistics of the U.S. Bureau of the Census in 1914 (Figure 6-1). Indian deaths for the registration areas were reported by age group



**FIGURE 6-1. Expansion of the registration areas for deaths (Bureau of the Census 1930:7).**

and sex but did not include data on cause of death (Bureau of the Census 1906). The first year in which the Census Bureau's Vital Statistics report included information on Indian deaths by cause was 1906 for tribes in South Dakota, when the Cheyenne River, Pine Ridge, and Rosebud Indian reservations were counted, along with the Crow Creek and Lower Brule Indian reservations as a part of their larger counties. Otherwise, mortality statistics prior to 1921 rarely separated Native American deaths by cause, but rather grouped Native Americans into a larger grouping of nonwhite populations, called "Colored".

Virtually no health data were collected systematically for the Prairie Band Potawatomi during the late 19<sup>th</sup> and early 20<sup>th</sup> centuries. Snippets of information can only be culled from the



general correspondence of the Potawatomi Agency during this time. The reports of the commissioner of Indian affairs were of little use for evaluating the Potawatomi after 1906, since they omitted the agents' reports and revealed nothing regarding the conditions of the Potawatomi and other tribes. This change in the nature of the reports began with the 1904-1909 tenure of Commissioner Francis Leupp (Connelley 1918).

Records from the National Archives and Records Administration provided data from a number of sources. The records were categorized under the *Records of the Bureau of Indian Affairs, Record Group 75 1793-1989*, and were specifically located under 75.19.47 – *Records of the Horton Indian Agency, KS*. The *Letters of the Potawatomi Agency, 1899-1933* provided anecdotal information on illnesses, vaccinations, health initiatives, payments, sanatorium transfers, food allocations, physician/nurse requests, and epidemics. These letters mostly detailed issues regarding the administration of the Potawatomi reservation as well as other nearby sites under BIA jurisdiction. A few of them discussed the health of the Potawatomi, but detailed health statistics were most often lacking. The *Potawatomi Indian Agency Annual Statistical Reports on Health (1931-1936)* included a death schedule by name and age for 1931-1934, health conditions and deaths by cause in 1935 and deaths by date in 1936. Also among these papers was a relatively comprehensive listing of deaths from 1924-1933 that included information on age, sex and cause of death. *Records from the General Survey of the Potawatomi Sub Agency*, conducted by Haskell Institute in April 1931, contained some data on health conditions. Most of these data consisted of counts, in tabular form, of Indian blood quantum, children in schools, English speakers, church affiliations, income, household characteristics, and types of illnesses. These data were not extensive and not sufficient for significant statistical analyses; therefore, the tabular counts and frequencies alone are presented.

## **Results**

### **Letters of the Potawatomi Agency, 1899-1933**

The earliest references to health issues in these letters are restricted to smallpox. In January 1899 U.S. Indian Agent G.W. James authorized the distribution of 1,125 vaccine points to the Potawatomi Indian Agency. In December of that same year, enough smallpox vaccine to vaccinate 500 persons was delivered, but a February 15, 1900, letter reported a smallpox death notwithstanding. By September of 1901, the Potawatomi agency reported a severe epidemic of smallpox with nine cases, four deaths, and 93 children quarantined in the school. In addition to the information on smallpox provided by the Potawatomi Agency, Kansas historian W.E. Connelly (1918) reported epidemics of measles and whooping cough on the reservation in 1888.

Tuberculosis became the primary disease concern early in the 20<sup>th</sup> century. The 1904 Report of the Commissioner of Indian Affairs cited a reduction in the number of Potawatomi living on the reservation and an excess of deaths over births for a number of years; tuberculosis was named as the cause of this trend (Connelley 1918). A lack of any communication referring to health conditions occurs between 1904 and August 1914, when T.J. Scott, clerk in charge at the Potawatomi Agency, requested that two patients be admitted to the tuberculosis sanatorium at the U.S. Industrial School in Phoenix, Arizona. In 1919 another letter to the commissioner of Indian affairs reported that two girls were to be sent to the Sac and Fox sanatorium in Toledo, Ohio. However, a letter written in 1922 by Potawatomi Indian agency superintendent A.R. Snyder, reporting to Charles H. Lerrigo, executive secretary, Kansas State Tuberculosis Association, refers to an apparent decrease in tuberculosis since 1913, when four TB deaths occurred among the Potawatomi. Calendar year 1921 showed only two deaths. Snyder pointed out that every year since 1914 had shown a small increase in population but still only four deaths

from tuberculosis were recorded for 1921 among all four tribes (Potawatomi, Kickapoo, Sac and Fox, Iowa); total population, 1,500. Snyder then went on to state that there was very little tuberculosis among the Kansas Indians because the Indians observed the recommendations for ventilation and cleanliness that prevented tuberculosis. The Indians, he wrote, had the benefit of public school instruction, pamphlets and reading materials from the State Board of Health, and thus no other special programs or efforts were needed. Finally, Snyder reported that the tuberculosis prevalence among the Indian population was as low as it was anywhere in Kansas. Strangely, there is no mention of the impact of the Spanish flu on the reservation from the letters of this time. It is possible that the agency closed down during this time, as did many offices and organizations (Barry 2005).

The need for a regular physician at the Potawatomi agency is indicated in the letters. A letter dated December 1915 reported to the commissioner of Indian affairs that the Potawatomi agency had been without a physician for several years. Another letter from 1922 makes the same complaint. It is unclear whether a physician was sent to the Potawatomi agency between 1915 and 1922.

Letters written between 1922 and 1925 indicate outbreaks of some minor illnesses and related directives. A “near epidemic” of scabies, an infectious skin condition caused by the itch mite *Sarcoptes scabiei*, was reported in a letter from 1922. Superintendent Snyder reports suffering from the condition himself. A case of “well developed” goiter was reported in 1923, as were two more cases that were less acute. Interestingly, in 1925 Snyder sent out a directive for agency employees and their families to avoid using cigarettes, which Snyder derided as a terrible habit and detrimental to health; however, there is no indication that the directive applied to the Indians of the agency.

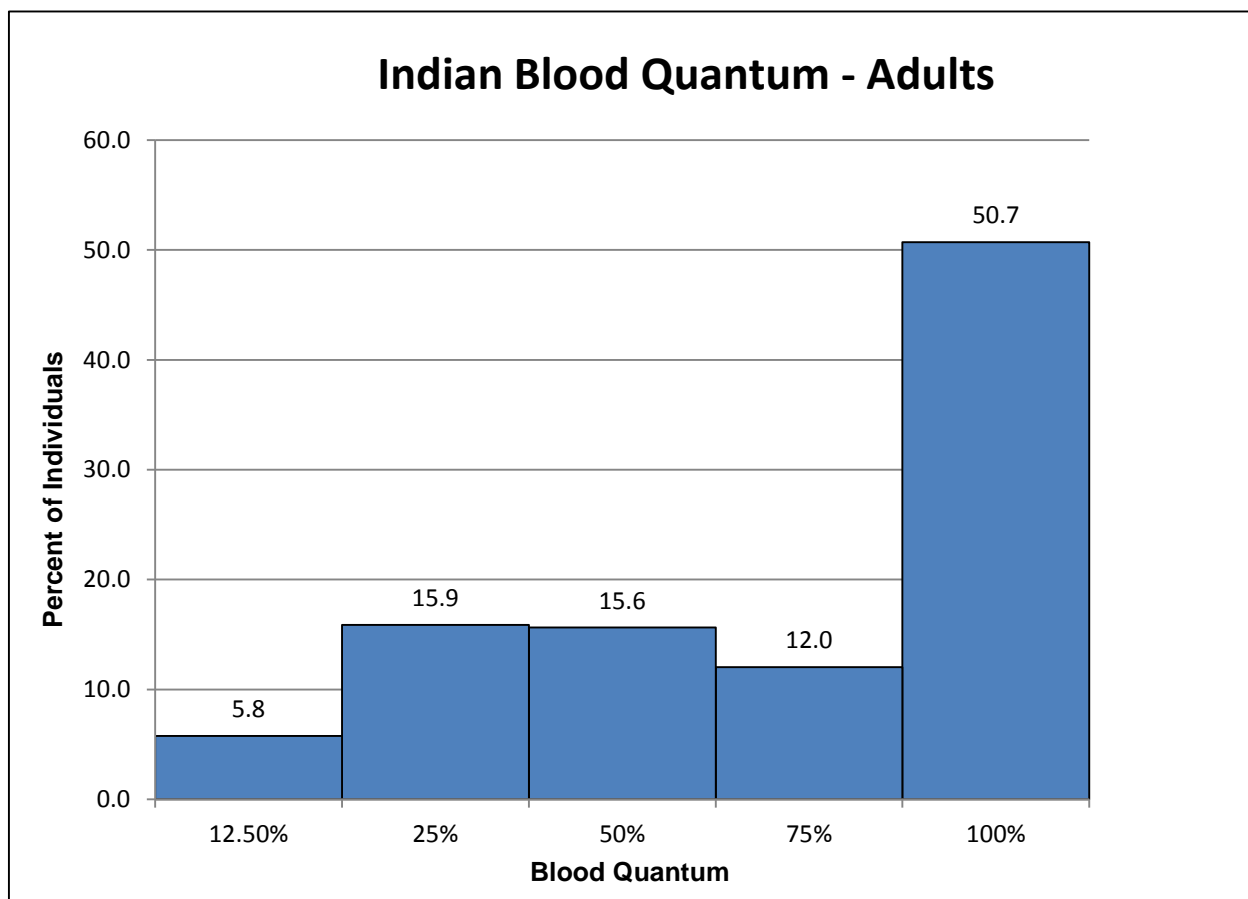
More serious illnesses were indicated in letters dated 1927 and after. In a May 9, 1927, letter from Potawatomi agency clerk Lloyd LaMotte to Superintendent C.M. Blair in Lawrence regarding payment for smallpox vaccine, LaMotte stated that the agency had experienced a serious epidemic of the disease during the past winter. On March 13, 1928, Special Physician J.L. Goodwin submitted a report to Dr. W.S. Stevens, the medical director in Shawnee, Oklahoma, indicating that he had examined 43 patients from the Potawatomi reservation. Most of the conditions described were problems with the eyes and included trachoma, conjunctivitis, pterygium, chalazion, entropion, and cataracts. Also listed were tuberculosis, goiter, and catarrhal deafness (Table 6-1). In early 1929 Potawatomi agency clerk Jasper Cross reported to George Peters in Lawrence concerning an influenza epidemic among the Indians of the jurisdiction, in which fully 60% of the Indian population on the reservation contracted the flu. In 1930 the Shawnee Indian sanatorium accepted seven people from the Potawatomi sub agency, considered accepting another following lab results, and rejected a two-year-old child.

**TABLE 6-1. Illnesses diagnosed by Special Physician J.L. Goodwin among 43 Prairie Band Potawatomi tribal members in March 1928.**

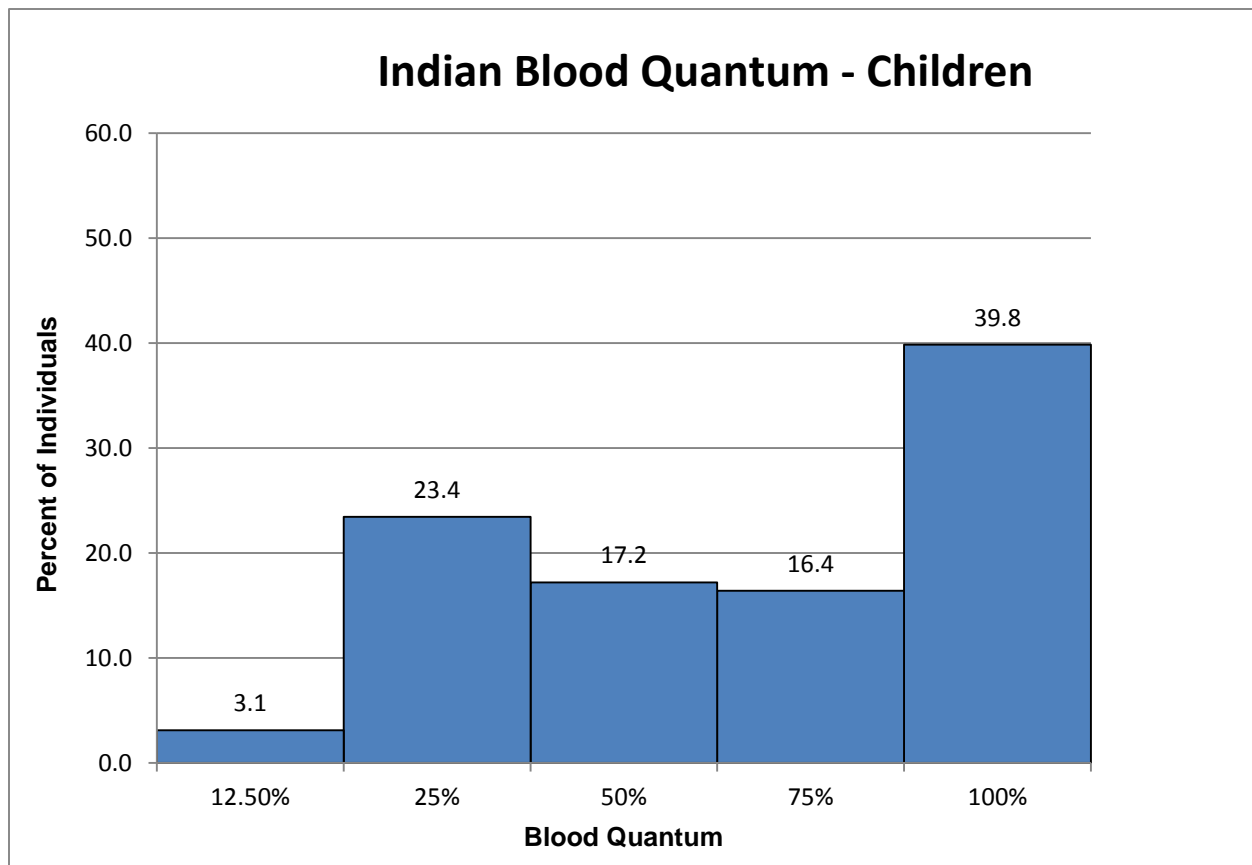
CONDITION	NUMBER
Trachoma	10
Conjunctivitis	4
Pterygium	2
Cataract	2
Catarrhal Deafness	1
Chalazion	1
Entropion	1
Goiter	1
Pyorrhea	1
Tuberculosis	1

## General Survey of the Potawatomi Sub Agency, Haskell Institute, April 1931

The general survey compared the living conditions of the Potawatomi with those of the Kickapoo Reservation, located approximately 40 miles north near Horton, Kansas. It was primarily a demographic report, not strictly a health survey. The unit of analysis was most often the household, with no specific count for the total number of individuals given. In general, conditions were better among the Potawatomi than among the Kickapoo. The survey noted that the Potawatomi reservation had both a Catholic and Protestant mission, likely as indicators of assimilation.



**FIGURE 6-2. Indian blood quantum among adult Potawatomi, 1931.**



**FIGURE 6-3. Indian blood quantum among Potawatomi children, 1931.**

The survey recorded only a few instances in which more than one family lived in a single home, which was reported as unsatisfactory living conditions. Of 204 Potawatomi families, only three lived in homes of one room, while 89 lived in homes of more than three rooms. The Potawatomi homes were described as being in a better state of repair and better furnished than those of the Kickapoo to the north. The homes in general were described as reasonably adequate

The principal source of family income was wage labor of various kinds and land rental, but such sources did not generally provide more than \$50 annually per family, although the Potawatomi apparently earned more annually per family than did the Kickapoo. Of the 204 Potawatomi families on the reservation, 37 were dependent solely upon farming for their livelihood. Some Potawatomi leased their land to white farmers in the area, for an average of

**TABLE 6-2. Personal Property owned by Potawatomi households as reported by the 1931 General Survey.**

Item	Families Having	Percent*
Chickens	67	33.0%
Horses	62	30.0%
Cows	35	17.0%
Hogs	29	14.0%
Sheep	0	0.0%
Gardens	133	65.0%
Autos	82	40.0%
Implements	76	37.0%
Trucks	5	2.5%
Tractors	1	0.5%

\* N=204 households

\$1.96 per acre annually. Forty percent of Potawatomi families owned cars, 30% owned horses, and 17% had cows (Table 6-2). One-hundred-and-thirty-three out of 204 of the Potawatomi families were reported to have gardens (65%), and the percentage of Potawatomi families experiencing inadequate food (31%, N=63) was reported to be lower than for Kickapoo families.

The survey was conducted during the time at which boarding schools were a primary means for indoctrinating children into the larger U.S. culture. Many children of the Potawatomi attended these off-reservation boarding schools. Haskell, Genoa in Nebraska, and Chilocco in Oklahoma were the boarding schools mentioned in the agency letters. Nearly 30% of school-aged children were sent to such schools, whereas the remainder attended the public schools in the region. Forty families were identified as unable to send their children to school due to financial reasons. Illiteracy was higher among Potawatomi adults than in other tribes: twenty-six (12%) adults were recorded as incapable of speaking English, while 65 (31%) could neither read nor write. These older Prairie Band tribal members represented the last generation that would speak Potawatomi as their first language.

The general survey listed a suspiciously high number of “retarded” children among the

Potawatomi: 108 children were so classified. Compared to the number of total children listed in or out of school (320), this suggested that 25%-34% of Potawatomi children were mentally handicapped, depending on how the surveyors quantified “total” children. Such a high percentage certainly reflects racist attitudes toward the Potawatomi children; other cases of illiteracy, emotional disturbances, congenital physical anomalies and/or chronic nutritional deficiencies may have been included. How children were determined to be retarded was not recorded.

**TABLE 6-3. Medical cases reported in the 1931 General Survey.**

<b>Condition</b>	<b># of cases</b>
Eye problems	9
Tuberculosis	6
Minor illness	5
Lung trouble	4
Syphilis	4
Tonsillitis	4
Bad teeth	3
Sickly	3
Heart problems	2
Blind	1
Kidney trouble	1
Paralysis	1
Rheumatism	1
Skin disease	1
Stomach trouble	1
Weakly	1

**TABLE 6-4. Causes of death among Potawatomi at the Potawatomi Agency, 1924-1933.**

<b>Causes</b>	<b>Number</b>	<b>Percent</b>
Accidents	3	2.9
Chronic diseases	15	14.3
Complications of pregnancy, childbirth, and the puerperium	1	1.0
Diseases of infancy	8	7.6
Infectious diseases	46	43.8
Other	33	31.4



Eye problems were the most common medical complaint in the general survey, followed by tuberculosis (Table 6-3). Unspecified “minor illnesses”—lung conditions, syphilis, tonsillitis, bad teeth and general “sickliness” were more prevalent than heart problems. The general survey did not contain data on mortality, but archival data separate from the general survey contained a listing of causes of death among Potawatomi of the Potawatomi Agency for the years before and after the survey, from 1924 to 1933 (Table 6-4).

A “Plan for the Development of an Extension Program on the Potawatomi Reservation” was developed by the superintendent of the Haskell Agency as a part of the general survey. Among the primary objectives of the plan was to stimulate community organizations so the home and family would be a part of a community rather than a socially isolated unit. A home extension program was to be established with the following objectives: to improve the care and feeding of all people on the reservation as well as children in boarding schools; to induce each Indian family to live in a separate house; to organize programs in community hygiene on water

**TABLE 6-5. Incidence of specific health conditions, Prairie Band Potawatomi Reservation, 1935.**

	<b>Total</b>	<b>Male</b>	<b>Male %</b>	<b>Female</b>	<b>Female %</b>
Gonorrhea	9	8	88.9	1	11.1
Syphilis	3	2	66.7	1	33.3
Chicken Pox	3	2	66.7	1	33.3
Impetigo	15	9	60.0	6	40.0
Influenza	98	33	33.7	65	66.3
Measles	84	38	45.2	46	54.8
Scabies	7	4	57.1	3	42.9
Tinea	4	3	75.0	1	25.0
Trachoma	3	1	33.3	2	66.7
TB	8	2	25.0	6	75.0
Trench Mouth	3	1	33.3	2	66.7
Whooping Cough	7	4	57.1	3	42.9
<b>TOTAL</b>	<b>244</b>	<b>107</b>	<b>43.9</b>	<b>137</b>	<b>56.1</b>

**TABLE 6-6. Health care and selected health statistics for the Prairie Band Potawatomi reservation, 1935.**

	Male	Female	Total
1. Live Births	14	14	28
Attended by Physician/Nurse	7	9	16
No Medical Attendant	7	5	12
2. Number of Stillbirths	0	1	1
Attended by Physician/Nurse	0	1	1
No Medical Attendant	0	0	0
3. No. of deaths, exclusive of stillbirths	5	10	15
Attended by Physician/Nurse	3	10	13
No Medical Attendant	2	0	2
4. No. Indians on Reservation who have:			
TB	3	9	12
Trachoma	25	10	35
Syphilis	14	6	20
Other VD	12	4	16
5. No. examinations for:			
TB	10	32	42
Trachoma	37	20	57
Syphilis	40	30	70
Other VD	19	5	24
6. No. of Positive cases for:			
TB	2	8	10
Trachoma	11	9	20
Syphilis	8	6	14
Other VD	10	1	11
7. New Cases of:			
TB	2	6	8
Trachoma	1	2	3
Syphilis	2	1	3
Other VD	8	1	9

supply, rubbish disposal, and quarantines; to improve the family food supply by providing for more gardens, fruit, poultry flocks, honey production, and greater milk and meat supplies; to help the Potawatomi recognize the importance of an adequate and safe water supply for every family, and to raise the standard of personal and domestic cleanliness; and to promote 4H Club work.

An Agricultural Extension Program of Work was also developed to improve agricultural conditions for the Potawatomi. It mainly focused on how a family farm was to be operated.

## Health of Potawatomi in 1935: The Potawatomi Agency Report

The 1935 Potawatomi Agency report included more detailed information about diseases on the reservation, reflecting a greater awareness of the need for and emphasis on health statistics. Unfortunately, the agency did not report the total number of individuals living on the reservation at that time, so it was not possible to calculate prevalence. Influenza and pneumonia dominated morbidity at the time (Table 6-5). Impetigo, TB, scabies, and whooping cough were also common pathologies. Births, deaths, and prevalence and incidence of TB, trachoma, syphilis and other venereal diseases are presented in Table 6-6. Males suffered from syphilis and other venereal diseases more than females. Females suffered more often from TB. One stillbirth was recorded out of 29 births (3.4%). Twice as many females died as males in 1935. Deaths were most commonly due to TB and pneumonia (Table 6-7). Mean age at death was 35 years and median age at death was 23, reflecting a number of deaths occurring among children less than 5 years old.

**TABLE 6-7. Causes of death on the Prairie Band Potawatomi Reservation, 1935.**

Age at Death, yrs	Cause of Death	Attended by Physician/Nurse
0.04	Premature birth	no
0.08	Pneumonia	yes
0.17	Peritonitis	yes
2	Tuberculosis	yes
2	Accident	yes
4	Tuberculosis	yes
18	Typhoid Fever	yes
23	Tuberculosis	yes
51	Pneumonia, Heart Problems, Hypertension, Syphilis	yes
59	Tuberculosis	yes
65	Colitis	no
69	Colitis	yes
75	Unknown	yes
80	Lung problems	yes
81	Pneumonia	yes

## Discussion

### Illnesses

Smallpox was a major health problem for Indians during the first part of the 20<sup>th</sup> century (Carter 1916), and the earliest letters from the Potawatomi Agency confirm that smallpox was a concern on the Prairie Band reservation at this time; it was still a major cause of morbidity as late as 1927. Tuberculosis, measles, and whooping cough were also cited. Actual numbers were rarely provided in reports of infectious disease outbreaks, aside from observations that “many” were affected. The predominance of such infectious diseases is consistent with information for other Indian populations at the time (see Chapter 5). Chronic conditions, on the other hand, were not indicated. Diabetes was not mentioned in the Potawatomi Agency letters of the early part of the century, and heart disease was much less prevalent than it would later become.

Potawatomi elders cited pneumonia, influenza, chicken pox, smallpox, mumps, measles, and particularly tuberculosis as being the common diseases of the day. One elder reported having a sibling who died of smallpox. Some elders remembered pneumonia as being among the most common causes of death. Diseases such as pneumonia and tuberculosis would have been particularly deadly to individuals already immunologically compromised by a previous infection such as measles and malnutrition.

As indicated by these various sources, many of the health data on the Prairie Band Potawatomi Reservation from the early part of the 20<sup>th</sup> century consisted of anecdotal descriptions in letters or reports from agency superintendents and other employees. Tables with actual counts by illness categories were scarce. These data and information are consistent with a population yet to complete the transition into the *Age of Degenerative and Man-Made Diseases*.

Infectious diseases accounted for over 43% of all deaths, whereas chronic conditions accounted for only 14%.

In addition to a lack of understanding of the importance of vital statistics, the dearth of accurate or consistent data collection during the early part of the century may reflect a desire on the part of Indian agents to downplay poor reservation conditions. Mitchell (1995), for instance, cites tuberculosis as a common cause of death among the Potawatomi in the 1930s, contradicting the Potawatomi Agency letters. Acknowledging the poor health and living conditions would have required the agents to recognize that the imposition of a new lifestyle was contributing to the mortality of the people under their care. Such was not the primary concern of the Indian agents and superintendents whose charge was to promote acculturation. Agents looking to remain in and advance in the government service were motivated to report successes and downplay problems (Prucha 1984).

## **Diet**

The 1931 general survey among the Prairie Band reported that 63 families, approximately 31% of the families surveyed, experienced food shortages, suggesting that for many individuals food insecurity was common two to four generations ago. Letters from the Potawatomi Agency in the 1930s list the most common commodities as potatoes, canned beef, rice, flour, pork, clothing, seed, chicks, and beef cattle. There also is some indication that the Agency had requested Red Cross food relief. A general BIA purchase order from 1932 is presented in Table 6-8. The most significant orders were for meats, salt, cornmeal, and lard.

During the Great Depression, many Prairie Band hunted wild animals and sold skins for money, while others planted gardens for home consumption as well as for a small surplus that could be sold (Figure 6-4). The Dust Bowl droughts of the 1930s were also difficult for the

**TABLE 6-8. General Purchase Order of the Potawatami Agency in 1932.**

<b>Articles</b>	<b>Quantity</b>	<b>Unit Price</b>	<b>Total Cost</b>
Beef	10 lbs	0.1	\$1.00
Boil Beef	10 lbs	0.1	\$1.00
Lard	16 lbs	0.0625	\$1.00
Meat	11 lbs	0.08	\$0.88
Pork	10 lbs	0.08	\$0.80
Coffee	3 lbs	0.23	\$0.69
Tea	1 lb	0.5	\$0.50
Sugar	10 lbs	0.05	\$0.50
Cornmeal	20 lbs	0.02	\$0.40
Salt	25 lbs	0.014	\$0.35
Kerosene	3 gals	0.11	\$0.33
Tobacco	1 lb	0.5	\$0.25
Crackers	2 lbs	0.125	\$0.25
Baking Powder	25 ozs	0.01	\$0.25
Macaroni	3 lbs	0.0833	\$0.25
Apples	2/2 lbs	0.05	\$0.11

Prairie Band. Few farms based on cash crops or livestock herds survived, whereas many tribal members persisted through subsistence farming of a wide variety of crops, as well as keeping chickens and milk cows. Hunting wild game also contributed to subsistence (Mitchell 1995).

Despite the findings of the 1931 general survey, most Potawatomi were able to secure adequate amounts of food. Though reported by some families, food scarcity apparently was not the predominant health concern on the Prairie Band reservation at this time. Contemporary Potawatomi elders report that they usually had plenty to eat, contradicting the general survey. One elder even provided the commentary “we were always on the chunky side.” Three meals a day was common, with only a few households limited to two meals a day; only one elder reported ever going to bed hungry. Food shortages were most likely in winter months, primarily due to the seasonality of plant staples. Those who reported being poor when young (most of the elders) recalled “living off the land.” According to these elders, hunting was common, and the primary game were squirrels, rabbits, fish, turtles, pheasant, and quail. Fishing was also

common. Virtually all recalled keeping gardens when growing up. Plants grown included corn, potatoes, beans (green), onions, lettuce, tomatoes, cucumbers, pumpkins, melons, squash, and peas. One Potawatomi elder reported that her grandmother grew traditional medicines in her garden. The tribal elders who lived on farms as children also had hogs, chickens, and cows as sources of food. Wild foods were commonly collected and included raspberries, gooseberries, milkweeds, strawberries, and mushrooms.

Many foods were preserved for use out of season. As children, Potawatomi elders reported storing and preserving garden produce in tin cans (canning). The tins could be placed in springs and streams to keep them cool, or simply left outside during the winter months. Fruit



**FIGURE 6-4. (Left) Potawatomi men butchering a deer on the Prairie Band Reservation, 1930s; (Right) 1930s view of a Prairie Band Potawatomi wood frame building and a garden plot with an arched branch framework. (courtesy Denver Public Library, Western History Collection).**

preserves (jellies) were also made. Some produce was dried, particularly corn and pumpkin. Interestingly, although growing food was common and important when these elders were young, few maintained gardens as they grew older. Among those who continued to garden, the size of plots and variety of plants grown were reduced.

Corn was a staple and was prepared in a variety of ways, including hominy, corn soup (*ndamnabo*), corn bread/corn cakes (*pugna*) and a parched corn cereal or mush called *kakazawabo*. Milkweed (*nInwezhe'k*) of the *Asclepias syriaca* variety was a popular wild food when in season, usually made into a soup (Figure 6-5). Fry bread (*saskokwe'te'k kweshken*) was commonly eaten at the time. Not a traditional food, Native Americans developed fry bread from



**FIGURE 6-5. Prairie Band elder identifies milkweed (*nInwezhe'k*), a traditional, wild plant food (KUMC RIT Program).**



the lard and flour that were common commodity items during the reservation period. High in calories but low in other nutrients, today fry bread is controversial among Native Americans due to its origins and its perceived contribution to obesity. At 700 calories, a serving of fry bread also supplies 53% of the RDA for fat, 67% of which is saturated, as well as 35 mg of cholesterol, 1274 mg of sodium and only 1.4 g of fiber (Berzok 2005; Harjo 2005; Mihesuah 2003, 2005; Wagner 2005).

Both the 1928 health survey and 1931 general survey cited goiter as a noninfectious condition that afflicted many Potawatomi. Not normally seen in the U.S. today, goiter is usually due to iodine deficiency. The condition was dramatically reduced following the introduction of iodized salt. Although there is no indication in the records of when the Prairie Band started using iodized salt, Table 6-8 lists salt as a common commodity, and iodized salt probably made its way into the Potawatomi diet after its use became widespread by the 1930s.

The mixed dietary strategy of hunting, gathering wild plants, and gardening supplemented the nutritionally inadequate and unsustainable agricultural schemes introduced by Euro-Americans. Thus, despite interference from the outside, the Potawatomi were able to develop a successful subsistence strategy that generally supplied them with the food they needed, in spite of development promoted by white society that was actually detrimental to Potawatomi well-being.

### **Activity**

Many Potawatomi elders reported that walking was the primary mode of travel when they were young. Though a number of families owned cars (40%) and/or horses (30%) with carriages or wagons (as indicated on the 1931 general survey), for most, particularly the young, walking was how they got from place to place on the reservation, including to and from school. In



**FIGURE 6-6. (Top) Potawatomi man collecting tree bark, circa 1930s; (Bottom) Potawatomi woman carrying reeds for baskets and mats (Courtesy Denver Public Library, Western History Collection).**

addition to walking, basic household labor required physical exertion (Figure 6-6). Water from wells had to be hauled in buckets from outdoor pumps. The prevalence of wood stoves for heating and cooking required chopping and hauling wood. Hunting, fishing, gathering wild foods, tending gardens, and working farms all required more physical exertion than that associated with food procurement and employment activities today.

## **Economic Status**

Most of the Potawatomi lived in poverty throughout much of the 20th century—a consequence of the allotment process that had begun at the end of the 19th century and had such a negative impact on the economic well-being of the tribe. They had little money to show for leasing or selling their land, as prices were often well below the actual value. Farming and land ownership brought few economic gains to the Prairie Band. At the beginning of the 20<sup>th</sup> century, most Potawatomi not only lived in extreme poverty but they likewise were unable to improve their economic situation. Economic opportunities were few, and many tribal members sold their lands and left the reservation to seek work elsewhere (Mitchell 1995). Nearly half (48%) of all the families in the 1928 survey were evaluated as falling into the “poor” category. The surveyors themselves concluded that well over half of the families were poor. During the 1930s it was reported that multiple Potawatomi families sometimes lived in single-family homes as a way of sharing costs, because the only jobs available to reservation residents were federal work projects (Landes 1963, 1970). Living Potawatomi elders recall a time when little or no work was available on the reservation. With nearly half of the households classified as poor, there is little doubt that economic deprivation was a factor impacting the health of the Potawatomi.

Potawatomi elders have described their homes in the first half of the century as old houses – some called them shacks – of three or four “rooms” with no electricity or indoor

plumbing. The “rooms” were often nothing more than sections of one big room with the kitchen in one corner and beds in the other. Bathroom facilities consisted of outhouses. Water was gathered from wells and springs using buckets. Wood-burning stoves were used for cooking and heating. Kerosene lanterns were the only source of light after dark.

Ironically, the “Plan for the Development of an Extension Program on the Potawatomi Reservation” developed by the superintendent of the Haskell Agency as part of the general survey was designed to stimulate community organizations so home and family would be a part of a “normal community group” rather than a socially isolated unit. Considering the assault on traditional tribal entities that had provided community cohesiveness, this restructuring of society in an economic setting of increasing impoverishment most likely exacerbated the conditions that promoted poor overall health. Already culturally and economically marginalized due to years of subjugation, racism, and unscrupulous manipulation by U.S. citizens and government, attempts to enculturate the Potawatomi into white society only increased the socioeconomic and cultural distance between them and the modernizing dominant culture.

## **Conclusion**

The morbidity and mortality patterns of the Potawatomi during this time were consistent with a population that had yet to undergo the epidemiologic transition to the *Age of Degenerative and Man-Made Diseases*. The predominance of infectious conditions and rarity of chronic conditions differed markedly from the mortality profile for the rest of the country (Table 6-9). Heart disease was mentioned by the Potawatomi Agency documents but was much less common than in the rest of the country, while cancers, strokes, kidneys diseases, and diabetes appear to have been rare or completely absent.

**TABLE 6-9. Top-ten causes of death in the U.S., all populations, 1935 (NCHS 2011).**

<b>RANK</b>	<b>CAUSE OF DEATH</b>
1	Diseases of the heart
2	Cancer and other malignant tumors
3	Pneumonia and influenza
4	Intracranial lesions of vascular origin (strokes)
5	Nephritis
6	Tuberculosis
7	Non-motor-vehicle accidents
8	Motor-vehicle accidents
9	Premature births
10	Diabetes mellitus

## **Chapter 6 References Cited**

Barry, J.M. 2005. *The Great Influenza: The Story of the Deadliest Pandemic in History*. London: Penguin Books.

Berzok, L.M. 2005. *Food in American History: American Indian Food*. Westport, CT: Greenwood Publishing Group, Inc.

Bureau of the Census. 1906. *Mortality Statistics 1900 TO 1904, Special Reports, Department of Commerce and Labor*. Washington, DC: U.S. Government Printing Office.

—. 1930. *Mortality Statistics 1928*. Department of Commerce. Washington, DC: U.S. Government Printing Office.

—. 1975. *Historical Statistics of the United States, Colonial Times to 1970, Bicentennial Edition, Part 2*. Washington, D.C.: U.S. Government Printing Office.

Carter, M. 1916. Smallpox on an Indian Reservation. *The American Journal of Nursing* 17(2): 112-117.

Connelley, W.E. 1918. The Prairie Band of Pottawatomie Indians. *Collections of the Kansas State Historical Society 1915-1918* 14:488-570.

Harjo, S.S. 2005. *My New Year's resolution: No more fat 'Indian' food*. Indian Country Today, 1/20/2005.

Hill, Edward E. 1981. *Guide to Records in the National Archives of the United States Relating to American Indians*. Washington DC: National Archives and Records Service, General Services Administration.

Landes, R. 1963. Potawatomi Medicine. *Transactions of the Kansas Academy of Science* 66(4): 553-599.

—. 1970. *The Prairie Potawatomi: Tradition and Ritual in the Twentieth Century*. Madison: University of Wisconsin Press.

Mihesuah, Devon A. 2003. Decolonizing Our Diets by Recovering Our Ancestors' Gardens. *American Indian Quarterly* 27(3/4):807-839.

—. 2005. *Recovering Our Ancestor's Gardens: Indigenous Recipes and Guide to Diet and Fitness*. Lincoln, NE: University of Nebraska Press.

Mitchell, G.E. 1995. *Stories of the Potawatomi People: From Early Days to Modern Times*. Originally published in the Topeka Capital Journal.  
<http://www.kansasheritage.org/pbp/books/mitch/mitchbuk.html>. January 18<sup>th</sup>, 2005.

NARA (National Archives and Records Administration). 2009. Guide to Archival Holdings at NARA's Central Plains Region (Kansas City). <http://www.archives.gov/central-plains/kansas-city/holdings/rg-050-099.html>. May 15<sup>th</sup>, 2009.

NCHS (National Center for Health Statistics). 2011. *Leading Causes of Death, 1900-1998*. [http://www.cdc.gov/nchs/data/dvs/lead1900\\_98.pdf](http://www.cdc.gov/nchs/data/dvs/lead1900_98.pdf). June 1<sup>st</sup>, 2011.

Prucha, Francis P. 1984. *The Great Father: The United States Government and the American Indians*, Vols. 1 and 2. Lincoln: University of Nebraska Press.

Wagner, A. 2005. *Icon or Hazard? The Great Debate Over Fry Bread*. Associated Press, 8/21/2005.

## **Chapter 7: Patterns of health and disease among Prairie Band Potawatomi in the early 20th Century. Part II. The Indian Health Survey, ca. 1928**

### **Introduction**

In 1928, then Superintendent of Indians C.M. Blair and Jasper Cross, agent of the Potawatomi Reservation, became convinced that the conditions on the reservation were not conducive to good health. In an attempt to substantiate their convictions, they commissioned the “Pottawatomie Indian Survey,” a house-to-house analysis of the diet, sanitary conditions, and health status of the people in each household on the reservation. The government agents requested that the Kansas State Board of Health (KSBH) carry out the collection of information and then make recommendations as to how best improve on the observed conditions.

Unlike the Records of the Horton Indian Agency (Chapter 6), the *Potawatomi Indian Health Survey, ca. 1928* consists of sufficient raw data for statistical analysis. Photostat copies of these surveys and the findings of the Kansas State Board of Health are housed at the National Archives in Washington, D.C. The forms are listed with the *Records of the Bureau of Indian Affairs (BIA), Record Group 75, 1793-1989*, and specifically catalogued under *Records of the Health Division, 75.14.4*. A blank copy of the form is shown in Appendix A. The survey was conducted by KSBH representatives between June 14 and September 8. They spent 26 days visiting approximately 125 households. The actual year the survey was taken was apparently not clear to the National Archives, as they listed the year as “ca. 1928,” and wrote “1929 - 1930?” on the forms. The actual year of the survey was determined by contacting a tribal member still living on the Potawatomi reservation who was listed on one of the forms as a 3-month-old. This individual was able to establish the year of the survey by providing a date of birth. Thus, it was determined that the survey was most likely conducted during the summer months of 1928. This

chapter is an analysis of the data collected on the 1928 health survey forms to determine the prevailing health conditions of the time and establish at what point along the epidemiologic transition the Prairie Band were situated at that time.

## **Methods**

### **Data**

The form utilized Roman numerals, numbers, and letters to differentiate survey questions. Those designations are indicated below in parentheses to refer to the specific question on the form. Names and ages of individuals in each household were recorded (Sections I, II, III, see Appendix A). In addition, the “percent of Indian blood” was recorded for the mother and father of the household, as was their status (living or deceased). Sex was not explicitly recorded on the survey forms, other than for people identified as the mother or father of the household. For this analysis sex was determined primarily by the gender most likely associated with the given name of the individual. In cases where the name was not indicative of one sex or the other, sex was not assigned. Other data collected during the survey were the primary occupation of the head of the family (Section IV); the occupations of other wage earners in the household (Section V); the number of people in the household who were able to read and write English (Section VI); and those in the household who could and could not speak English.

For this analysis, income groupings were developed from the short descriptions of “total annual income of family” (Section VII) recorded by the original surveyors. Sometimes surveyors entered an income, at other times they simply made a statement such as “meager” or “well-off.” Based on the various descriptive and/or monetary data recorded by the surveyors, income groups of “good,” “fair,” and “poor” were developed to determine what impact income might have on the other social and disease variables.



In some cases the surveyors met with the head of household or family somewhere other than at their home; thus, sanitary conditions were not evaluated for all households. For those that were, the surveyors simply checked “good,” “fair,” or “poor” for sanitary condition (Sections VIII 2 a, b, c), and often no other description was given. The number of beds in each home was recorded (Section VIII 3 a). Ventilation (Section VIII 3 b) generally was not described on the survey form, although it was noted in the summary. Use of individual towels among people in the household was recorded primarily on the form as a “yes” or “no” (Section VIII 4). Surveyors also noted the type and location of bathroom facilities in each of the households, whether these facilities were protected from flies, and if they might be contributing to soil pollution (Sections IX 1, 2).

Drinking water quality had to be determined by examining what the surveyors wrote with regard to the source of water, the location of the water source relative to contaminants and runoff, whether the water was protected from contamination, and whether the water had ever been tested (Sections X 1, 2, 3). Much of the pertinent information was recorded in the “Remarks” section under “Water Supply.” A determination of water quality as “good” or “poor” was made based on the information recorded by the surveyors, who most often used these terms.

Under “Family History” surveyors recorded the name or relationship, age, and cause of death of family members (Section XI). Current health problems, the sufferer's name and marital status were recorded for living members of the household (Section XII). Other health data included serious illnesses experienced in the past five years (Section XIII), any bouts with tuberculosis (Section XIV), and availability of medical (Section XV) services, as well as dental, nursing, and midwifery services (Section XVI).

Dietary intake data (Section XVII) were collected in the form of a listing of the "daily menu" for the household, although the survey was inconsistent in collecting these data across households. The frequency of food intake was usually recorded only for milk and meat. No attempts were made by the surveyors to record the amount of food eaten, so estimates of caloric intake were not possible. In some cases, the diet was only described as "good" or "general," with no indication of what, specifically, was eaten.

### **Statistical Methods**

Frequencies of health conditions and illnesses were tabulated from Section XII of the 1928 KSBH survey. These were conditions recorded by the surveyors at the time as having been most recently experienced by Potawatomi family members. The most commonly reported conditions were tuberculosis, pneumonia, measles, "chest problems," "heart problems," goiter, rheumatism, trachoma and "other eye problems." All other, less commonly reported conditions were summed per household. In addition, a variable was calculated that equaled the sum of the total number of diseases per household (each illness reported = 1), divided by the number of residents of the household, to create an index of disease prevalence that is independent of household size.

For the purposes of analysis, groupings were developed based on whether the household had an adequate or inadequate supply of particular categories of foods (meat, milk, fruits, or vegetables). The availability of milk and meat were recorded, as well as the frequency of their consumption. For milk consumption, households generally either had access to milk or did not. Thus, two groups were constructed based on milk intake: "available" or "unavailable." Meat intake was classified as "frequent," "intermediate," or "seldom." Households that had meat everyday were placed in the "frequent" category; those that reported having meat at least once a

week to every other day were placed in the “intermediate” category; and those who consumed meat less than once a week were placed in the “seldom” category. Fruit and vegetable intakes also were categorized as "available" or "unavailable," based on the data recorded for the daily menu. Macronutrient content (protein, fat, carbohydrate) of foods itemized in the daily menus, as a percentage of the overall contribution to caloric intake, was estimated using Mosby Nutritrac for Windows Version 1.0 (1998).

Univariate statistical methods were utilized to determine which variables were associated with or contributed to particular health conditions. Nonparametric correlation and basic descriptive statistics were the primary techniques used. Kendall’s coefficient of rank correlation (tau-b) was used to determine the relationships among variables and to establish what factors contributed significantly to the recorded health conditions. Sociocultural variables available for analysis were income class, household size (number of people living in the home), number of available beds per person for the home, water quality, and sanitary conditions.

Biological/demographic variables included Indian blood quantum (recorded as “percent of Indian blood,” a measure of the degree of Indian genetic ancestry), age, and sex. Disease conditions included in this analysis were tuberculosis, pneumonia, measles, chest problems, heart problems, goiter, trachoma, eye disorders, rheumatism and “other.” Diet was analyzed for correlations with other variables and was grouped into the macronutrients carbohydrates, fat, and protein. Some variables were strongly intercorrelated, and partial correlation coefficients were computed for these (see Results below).

Because much of the data are categorical, contained missing values and are non-parametric, they were first standardized by subtracting the mean from each observation and dividing by the standard deviation. Principal components analysis was then used to group the

data into a few dimensions by constructing a correlation matrix whose elements were the correlations that were determined by first running the data without missing values. The correlation matrix, rather than the raw data, was used for final analysis. Once the coefficients for the axes were determined, varimax rotation was used to maximize the calculations of scores on the axes. Variables that did not load on a particular axis were removed to calculate scores for more individual observations (Hill and Lewicki 2006). The structure was run several times to extract a variable number of factors until a best model was attained: a variation of the Kaiser Criterion was used to determine the number of factors to retain (Kaiser 1960). Rotated factors with eigenvalues greater than 1.5 were retained, meaning that a factor must explain at least 1.5 times the variance of one original variable. Variables with a communality of less than 0.1 were removed due to their having little impact on the model.

The variables created from factor scores were used in partial least squares regression (PLS). The PLS extends a multiple regression model by increasing the number of dependent variables that can be analyzed while avoiding issues of collinearity when using factor scores in a general linear model. It is expressed as:

$$Y = b_0 + b_1X_1 + b_2X_2 + \dots + b_pX_p$$

where  $b_0$  is the regression coefficient of the intercept and the  $b_i$  values are regression coefficients (for variables  $1$  through  $p$ ) computed from the data. Between-subject designs using ANOVA provided p-values to evaluate the model for each variable.

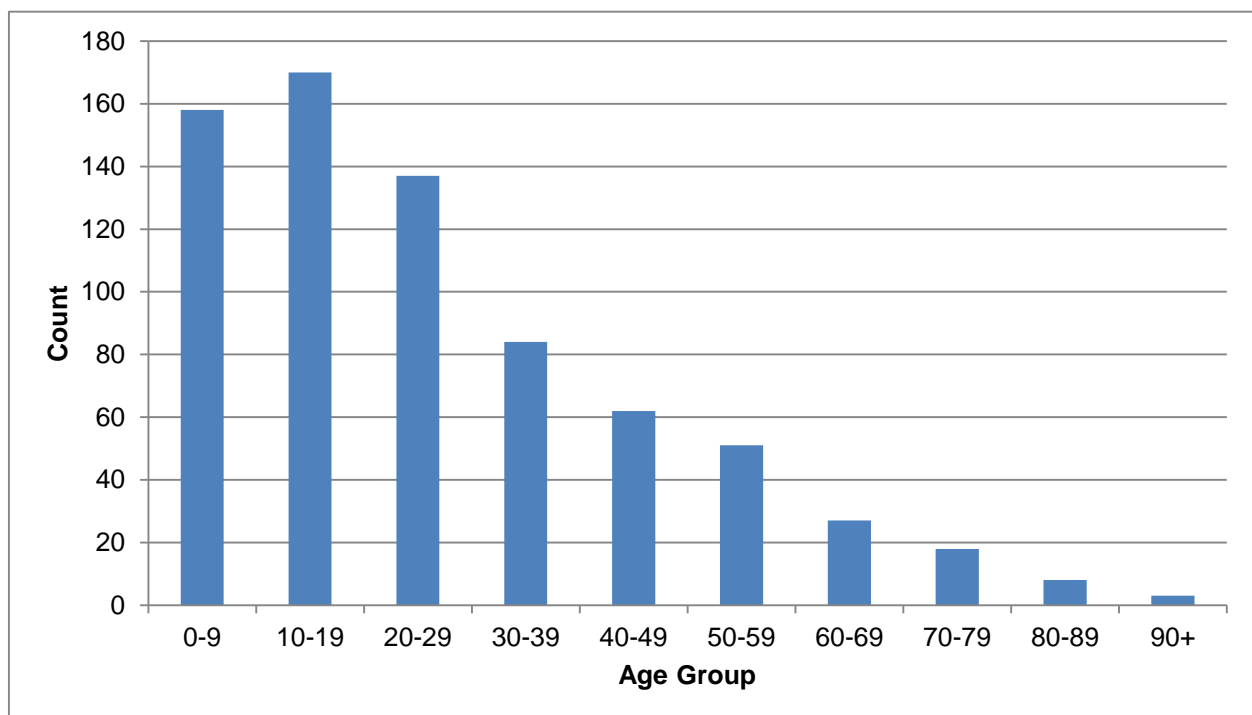
## Results

The Potawatomi Indian health survey of 1928 provided health information for 784 individuals (375 males and 364 females) from 125 households (Table 7-1). Sex could not be

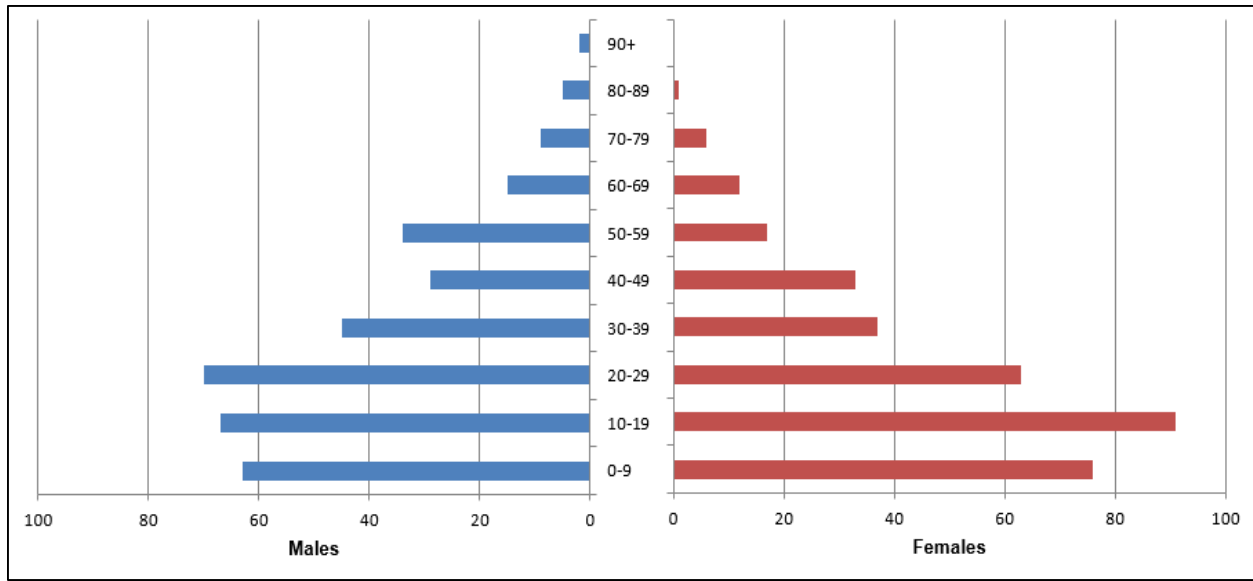
determined for 45 people. Mean age of the people surveyed was 26. Of the 445 children for which information was obtained (including adult children of the household's parents), only 342 were full-time residents on the reservation. Many attended off-reservation boarding schools such as Haskell Institute in Lawrence, Kansas, or Genoa Indian School in Genoa, Nebraska. Others had relocated off the reservation in search of work in cities. The mean number of people per

**TABLE 7-1. General demographics of the people surveyed in 1928.**

PARAMETER	STATISTIC		
Number of Households Surveyed	125.0		
Number of Living Individuals on the Survey	784.0		
Number of Males	375.0		
Number of Females	364.0		
Mean Age	26.2	±19.7	
Median Age	21.0		
Mean Number of People per Household	5.5	±2.7	
Mean Number of Rooms per Household	4.1	±2.0	
Mean Number of Beds per Household	2.6	±1.3	



**FIGURE 7-1. Age distribution among the Prairie Band Potawatomi in 1928.**



**FIGURE 7-2. Population pyramid for the Prairie Band Potawatomi in 1928.**

household was 5.5. The average house had just over four rooms and only 2.6 beds. One home had a sleeping porch. “Beds” included regular beds, cots, hammocks, or davenports. Given the number of people per household and the average number of beds, it is obvious that some people either had no bed on which to sleep, or had to share beds with others. The surveyors reported that a number of people in several households slept on the floor. The mean age of 26.2 indicates a young population (Figure 7-1), and a population pyramid is shown in Figure 7-2.

The majority of Potawatomi heads of household identified farming as their primary occupation (Table 7-2). The next most common livelihood was to rent land to white farmers and ranchers. KSBH Surveyors recorded some information on family income (Table 7-3). Income was described as “good” for 32 of the households, “fair” for 23, and “poor” for 51 of them. In their summary, the surveyors reported that over half the Potawatomi considered themselves poor and that the average annual income of these 73 families was approximately \$100. The surveyors characterized four families as being “well fixed,” and 48 as having comfortable incomes.

Unfortunately, since this information is given as a part of the summary, there is no indication as to which families fell into which categories. Nor is this information consistent with how data were recorded on the survey forms.

### Sanitary Conditions

Little information is provided as to the criteria the surveyors used for making their determination on sanitary conditions. Evaluations were made for 96 of the homes. Seventeen were evaluated as “good,” 35 as “fair,” and 44 as “poor” (Table 7-4). The KSBH surveyors made note of the quality of drinking water in each household. The majority of the households obtained water from wells (Table 7-5), most of which were hand-dug. Only two families had running water in the home. Six households utilized natural springs for water. One household

**TABLE 7-2. Occupations in 1928.**

<b>Occupation of Head of Household</b>	<b>Number</b>	<b>Percent</b>	<b>Occupation of Others</b>	<b>Number</b>	<b>Percent*</b>
Farming	79	63	Farming	8	42
Rent Land	23	18	Rent Land	3	16
Labor	17	14	Labor	7	37
Ranch Hand	2	1.6	Ranch Hand	1	5
Trucking	2	1.6			
Carpentry	1	1			
Agency	1	1			

\*Percent based on 19 total people.

**TABLE 7-3. Income groups, 1928, based on surveyors' summaries**

<b>Group</b>	<b>Number</b>	<b>Percent*</b>	<b>Summary</b>
Good	32	30	4 "well fixed"
Fair	23	22	48 "comfortable incomes"
Poor	51	48	73 "poor"

\*Percents based only on those households in which an income group could be assigned (n = 106).

**TABLE 7-4. Sanitary condition of housing in 1928.**

<b>Condition</b>	<b>Number</b>	<b>Percent*</b>
Good	17	18
Fair	35	37
Poor	44	46

\*Percent based only on those households in which a sanitary condition was determined (n = 96).

**TABLE 7-5. Household water sources.**

<b>Source</b>	<b>Number</b>	<b>Percent of Total</b>	<b>Water Quality*</b>	
			<b>Good</b>	<b>Poor</b>
Well	106	85	33	39
Spring	6	5	0	5
Plumbed	2	2	2	0
Cistern	1	1	1	0
			36	44

\*Water quality could only be determined for 80 homes, thus the totals for water quality for each source do not add up to the total by source.

utilized a cistern, but no details of the nature of the cistern were recorded; very likely it was nothing more than a pit dug to collect rainwater. The surveyors did note that there was no filter for the cistern. A number of households did not have their own source of water. Among these, several obtained water from their neighbors. Based on what the surveyors recorded, household water quality was described as either “adequate” or “poor.” Among those households in which water quality was determined, more than half had a poor supply of drinking water, most often due to either the lack of a cover to protect the well from contaminants or the location of the well. The surveyors criticized wells in low-lying areas for being vulnerable to water run-off and wells located in close proximity to barns or animal pens, for their risk of contamination by farm waste.

Also of interest to the surveyors was the nature and sanitary condition of the toilet facilities (Table 7-6). Ninety-eight (78%) of the households had outdoor privies. The surveyors recorded 16 “no” or “none” for the “Outside privies?” question. It is not immediately clear what



**TABLE 7-6. Toilet facilities in 1928.**

DESCRIPTOR	NUMBER	PERCENT OF TOTAL
Outside Privies	98	78
Pits Dug	82	66
Use Lime	12	10
Protected From Flies	11	9

this meant with regard to toilet facilities: whether they had indoor plumbed facilities or no facilities at all. Only two households reported having water piped into their homes, and both of these households had outdoor privies. Given the rural nature of the area and its lack of infrastructure, it is most likely that those having no outside privies had no toilet facilities, other than nature itself. It is likely that households in which nothing was recorded for “Outside privies?” lacked toilet facilities. Most (81) of the outhouse facilities were not protected from flies. Only 12 households reported the use of lime in their toilets. Eighty-one of the 125 households reported not having individual towels for each member of the home. Only two homes had individual towels for each member, and one home reported that one of its residents had his own towel. No answer to this question was obtained for forty-one households.

## **Mortality**

Individuals were surveyed as to the cause of death of deceased family members, which was reported for 370 individuals. These were usually parents, spouses, or children of the heads of household. It is possible that many of the deaths were counted more than once, since the names of the deceased were often not included; rather they were recorded as “maternal father,” for instance, for the death of the household mother’s father. Without genealogies of the families surveyed (which were not compiled), it is difficult to say how many siblings living in different households reported on the deaths of the same parents. Cause of death was unknown for over a

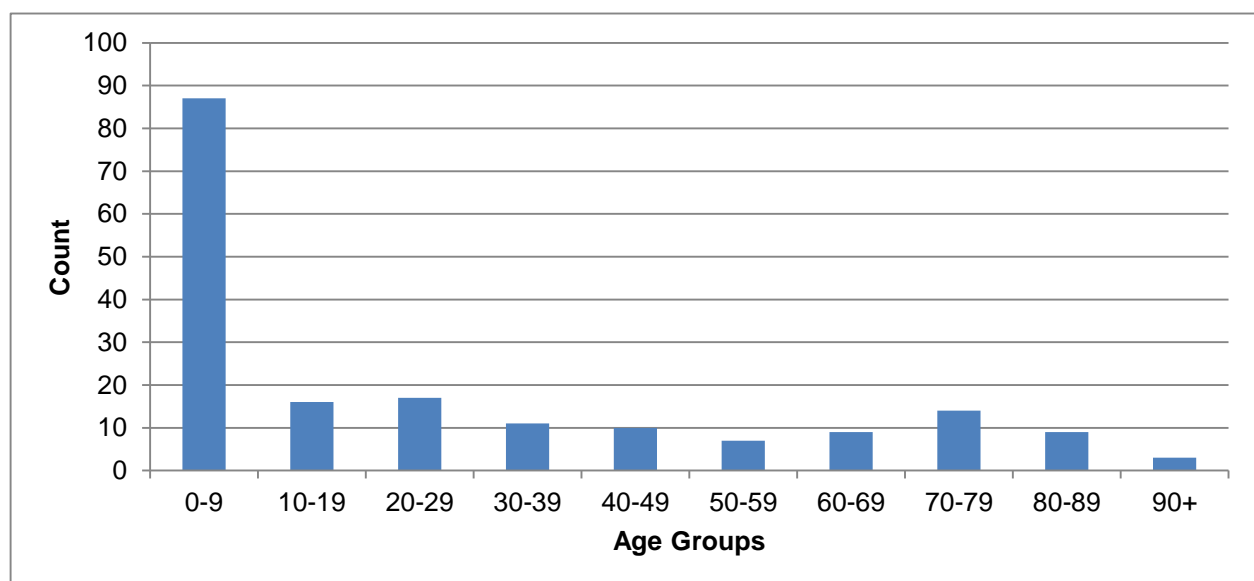
quarter of the deaths reported. Among those deaths to which a cause could be attributed, tuberculosis was most often cited, followed by pneumonia, accidents, childbirth, and heart problems (Table 7-7). Distinctions were not made as to the nature of the tuberculosis infections (lung, gland, bone, etc.), cause of accidents, or types of heart trouble. The mean age at death for those whose ages were reported was 25, indicating a large number of deaths at young ages (Figure 7-3).

As part of their summary and conclusions, the KSBH surveyors also discussed the number and causes of deaths among the Potawatomi for the years 1926 and 1927, although no

**TABLE 7-7. Five most often reported causes of death**

CAUSE	NUMBER	PERCENT OF CAUSES*
Tuberculosis	48	13
Pneumonia	43	12
Accidents	22	6
Childbirth	18	5
Heart Problems	15	4

\*Based on 369 deaths.



**FIGURE 7-3. Age distributions of deaths where age is reported.**

information was given as to where they collected this information. They recorded 28 deaths for these two years and listed the causes of death in order of their prevalence. The most common cause given was tuberculosis, followed by pneumonia, heart trouble, stillbirths, starvation, nephritis, suicide, and cancer. No figures are provided as to how many died from each cause, with the exception of stillbirths, of which there were nine cases, placing it seventh among causes of death listed on the survey forms.

The data from the forms and the summary are in agreement that tuberculosis and pneumonia were the most common causes of death among the Potawatomi at this time. Heart problems were also among the top five from both sources. Unique to the summary are deaths due to stillbirths and starvation. The data forms recorded stillbirths as “died at birth” and specify that it was the woman giving birth who died, not the infant. Neither “Starvation,” nor any similar cause of death was ever listed on the survey forms, so it is difficult to say anything substantial with regard to its incidence, other than to report what the surveyors wrote in their summary.

## **Morbidity**

Among the illnesses and health conditions reported for living members of the households (Table 7-8), eye disorders were most prevalent, accounting for almost a quarter of all reported conditions. This category consisted of people who reported having trachoma (3), sore eyes (8), blindness (8) and eye problems in general (23). Thirty-four (>27%) of the 125 households reported experiencing some form of eye problem. Enlarged thyroid gland or goiter was the next most often reported malady, followed by tuberculosis, heart problems, and rheumatism. Of the 24 people reportedly suffering from enlarged thyroid, 23 were female; the sex of the 24th person was not recorded on the survey and could not be assigned based on the first name. A number of

**TABLE 7-8. Five most often reported health problems in 1928.**

<b>Condition</b>	<b>Number</b>	<b>Percent of Conditions</b>	<b>INCIDENCE</b>
Eye Problems	44	24	5.6%
Enlarged Thyroid/Goiter	24	13	3.1%
Tuberculosis	19	10	2.4%
Heart Problems	14	8	1.8%
Rheumatism	12	7	1.5%

people reported having lung or chest problems, but no other information was provided as to their condition. Thus, it is difficult to say whether these conditions were due to tuberculosis, heart trouble, pneumonia, bronchitis, or several other illnesses that manifest these symptoms, perhaps leading to an underestimation of the frequency of these illnesses.

The prevalence of diseases reported by the surveyors in their summary was inconsistent with the data they recorded. In their summary, they reported 14 active cases of tuberculosis, 19 suspect cases and 27 cases to continue observing for symptoms. The summary listed 28 households as having incidence of enlarged thyroids, a few more cases than what they recorded on the forms. The number of people suffering from eye conditions is not given in the summary, although the condition was widespread. Despite the discrepancies in numbers for the first two conditions, there is consistency between the data and the summary in that they both indicate that eye problems, goiters, and tuberculosis were the most frequent pathologies among the Potawatomi at this time.

KSBH surveyors asked about the incidence of serious illness experienced in the households in the past five years. Tuberculosis was most often cited, followed by pneumonia and typhoid, then influenza and smallpox (Table 7-9). Occurrences of scarlet fever and Bright's disease, an often chronic inflammatory disease of the kidneys, were also reported. Data for this question in the survey were possibly inadequately recorded, given that the number of serious

**TABLE 7-9. Most common serious illnesses among households in the five previous years.**

<b>Condition</b>	<b>Number</b>	<b>Percent of Total</b>
Tuberculosis	9	32
Pneumonia	5	18
Typhoid	5	18
Influenza	3	11
Smallpox	3	11

**TABLE 7-10. Health Care Services utilized in 1928.**

<b>Service</b>	<b>Number</b>	<b>Percent*</b>
Physician	67	54
Midwife	67	54
Dental	44	35
Nursing	6	5

\*Based on the total number of households, 125.

tuberculosis cases (9) listed here is much fewer than the number of people suffering from tuberculosis (19) at the time of the survey.

The number of households that had utilized health services is shown in Table 7-10. Many Potawatomi did not have medical services available to them, since they had to pay for any health care services they received and the cost was prohibitive. Those who did seek medical assistance did so only out of extreme necessity. The categories of health care refer primarily to the profession of the individual providing services. Approximately 25% of households that reported having midwife services did not utilize this resource for every birth. Only about half of the households surveyed were able to utilize any health care services.

### **Diet and Nutrition**

The surveyors seemed most preoccupied with documenting three foods in particular: milk, butter, and meat. They also emphasized vegetables to a certain extent, particularly “greens.” They described the meals of the poorest Potawatomi as consisting of dried beans, rice,

potatoes, bread and syrup, with cabbage, corn, tomatoes, wild berries, and grapes available in the summer. Almost half of the households consumed little or no milk (Table 7-11). Reportedly, this was due primarily to its cost. Among the households that did have milk, 22 kept cows, and 18 bought milk on a regular basis. Several households reported having milk only “occasionally.” The surveyors state that the Indian children are “unusually fond” of milk. Whether this was evident to them from their survey or something they had heard about Indian children is not clear. Fewer than half of the families (59) consumed butter. Most of the families had at least a variable supply of meat. No indication of the source of meat, whether beef, pork, goat, or game, was given. Consumption of bacon and chicken was recorded, although very few families reported having either regularly. Only 16 of the households “seldom” ate meat. Most families had meat at least once a week, and many (28) had meat every day. Meat intake was not indicated for 24 households. Of these, only three have no dietary information whatsoever. The remaining 21 households simply may not have had any meat available to them, and the lack of meat was not recorded. Given the menu described by the surveyors for the poorest Potawatomi, this very likely was indeed the case. Just over half of the households consumed few or no vegetables. Few families ate “greens” or spinach. The most commonly consumed vegetables were cabbage, tomatoes, green beans, lettuce, onions, corn, and peas. The source of these vegetables was often

**TABLE 7-11. Dietary intake in 1928.**

INTAKE	NUMBER	PERCENT OF TOTAL
Milk Available	65	52
Milk Unavailable	60	48
Meat Intake Frequent	28	28*
Meat Intake Intermediate	57	56*
Meat Intake Seldom	16	16*
Vegetables Available	60	49
Fruits Available	19	15

\*Percents based only on those households in which meat intake was recorded (n = 101).

a household kitchen garden during summer; thus they were available only in season.

Consumption of fruit was even less frequent. Only 19 households had any access to fruit. Like vegetables, fruit was available only seasonally. Among those that did have fruit, consumption was reported as being occasional at best. Among the fruits eaten were gooseberries, wild berries, apples, grapes, and raisins.

No analysis was conducted with regard to the amount of food consumed. It is unlikely that the portions were very large. Given the foods recorded, it is possible, however, to calculate the proportions of macronutrients provided in the diet. The diet of the poorest Potawatomi (dried beans, rice, potatoes, bread, syrup, cabbage, corn, tomatoes, wild berries, grapes) as described by the surveyors would have been very high in carbohydrates, contributing close to 80% of the total caloric intake, with protein (~14%) and fats (~8%) making up only about 22% of the total (Table 7-12). As might be expected, carbohydrate and fat intake was correlated with income (Table 7-13). Fat intake increased and carbohydrate intake decreased among the higher income households. For all of the Potawatomi surveyed, mean carbohydrate intake as a percent of total energy intake is estimated to be 45.8%. Mean protein intake was an estimated 27.5% of total intake, and fat contributed 26.7%.

**TABLE 7-12. Macronutrient intake in 1928.**

	<b>Potawatomi (all)</b>	<b>Potawatomi (poorest)</b>	<b>Current Recommendations</b>
Carbohydrate	45.8%	82.4%	45-65%
Fat	26.7%	3.7%	20-35%
Protein	27.5%	13.9%	10-35%

## Correlations

The most highly significant correlations are shown in Table 7-13 through 7-16.

Pneumonia and measles had a high positive correlation with each other, and pneumonia sufferers tended to be younger. Total illnesses had high positive correlations with sanitary conditions and privies being protected from flies. Indian blood quantum was negatively correlated with income and sanitary conditions, the latter two also being highly positively correlated with each other. Measures of sanitary conditions correlated primarily with each other and contrasted with blood quantum and income variables. Income was additionally correlated with dietary intake.

Individual Indian blood quantum primarily followed the same patterns as those of the parents, as might be expected (Table 7-14). The percent of Indian blood of the father of the household was negatively correlated with income, sanitary conditions, and the presence of privy pits. The degree of Indian blood of fathers and mothers in the households was highly positively correlated. Mothers' degree of Indian blood was negatively correlated with income, water quality, and sanitary conditions. Sanitary conditions in turn were correlated with many variables, including income, Indian blood quantum, and total illnesses.

**TABLE 7-13. Most significant correlations with disease variables.**

Illnesses			
Variable	Correlate	Correlation	p-value
Pneumonia	Measles	0.469	<0.001
Total Illnesses	Protected from Flies	-0.201	<0.001
Total Illnesses	Sanitary Conditions	-0.196	<0.001



**TABLE 7-14. Most significant correlations with biological variables.**

<b>Biological Variables</b>			
<b>Variable</b>	<b>Correlate</b>	<b>Correlation</b>	<b>p-value</b>
Father % Indian	Mother % Indian	0.348	<0.001
Mother % Indian	Water Quality	-0.279	<0.001
Indian Blood Quantum	Income	-0.248	<0.001
Father % Indian	Pit Dug	-0.242	<0.001
Indian Blood Quantum	Sanitary Conditions	-0.227	<0.001
Mother % Indian	Income	-0.225	<0.001
Mother % Indian	Protected from Flies	0.223	<0.001
Indian Blood Quantum	Pit Dug	-0.222	<0.001
Father % Indian	Milk Consumption	-0.218	<0.001
Father % Indian	Income	-0.217	<0.001
Father % Indian	Sanitary Conditions	-0.213	<0.001
Mother % Indian	Pit Dug	-0.209	<0.001
Mother % Indian	Sanitary Conditions	-0.199	<0.001

\*Indian Blood Quantum was determined using Father % Indian and Mother % Indian and were thus highly correlated at 0.782 and 0.769, respectively.

Income and sanitary conditions were highly correlated with one another, and these were the variables most often correlated with the other variables (Table 7-15). Illnesses were generally not highly correlated with sociocultural variables, but sometimes were correlated with each other. Only the “total illnesses” variable, consisting of a sum of all the illnesses experienced by the household divided by the household size, was correlated with more than two sociocultural variables. Goiters were significantly correlated with Indian blood quantum and sex but the correlations were not particularly high (0.019 and -0.158, respectively).

Dietary intake patterns were correlated with income and parental blood quantum (Table 7-16). Higher fat and protein intake were associated with greater income, whereas higher carbohydrate intake was associated with lower income. Higher milk consumption was associated with lower Indian blood quantum levels, and boosted fat and protein intake.

**TABLE 7-15. Most significant correlations with environmental variables.**

Environmental Variables			
Variable	Correlate	Correlation	p-value
Sanitary Conditions	Income	0.460	<0.001
Household Size	Beds per Person	-0.307	<0.001
Water Quality	Mother % Indian	-0.279	<0.001
Income	Fat Intake	0.273	<0.001
Income	Indian Blood Quantum	-0.248	<0.001
Pit Dug	Father % Indian	-0.242	<0.001
Water Quality	Protected from Flies	-0.242	<0.001
Sanitary Conditions	Outhouse	0.235	<0.001
Income	Carbohydrate Intake	-0.233	<0.001
Sanitary Conditions	Indian Blood Quantum	-0.227	<0.001
Income	Mother % Indian	-0.225	<0.001
Protected from Flies	Mother % Indian	0.223	<0.001
Pit Dug	Indian Blood Quantum	-0.222	<0.001
Income	Father % Indian	-0.217	<0.001
Sanitary Conditions	Father % Indian	-0.213	<0.001
Sanitary Conditions	Protein Intake	0.210	<0.001
Pit Dug	Mother % Indian	-0.209	<0.001
Income	Pit Dug	0.201	<0.001
Protected from Flies	Total Illnesses	-0.201	<0.001
Sanitary Conditions	Mother % Indian	-0.199	<0.001
Sanitary Conditions	Total Illnesses	-0.196	<0.001
Sanitary Conditions	Lime Use	0.194	<0.001

\*Indian Blood Quantum was determined using Father % Indian and Mother % Indian and were thus highly correlated at 0.782 and 0.769, respectively.

A partial correlation controlling for the effect of income on fat intake and sanitary conditions shows that the relationship existed between the variables regardless of income. The same is true with regard to milk intake and father's degree of Indian ancestry, holding income constant. The relationship between the degree of Indian ancestry and sanitary conditions is still significant with the effects of income removed.

Table 7-17 shows the results of principal components factor analysis for the 1928 data. Fourteen variables were included in the analysis. Varimax rotation produced three factors with

**TABLE 7-16. Most significant correlations with dietary variables.**

<b>Dietary Variables</b>			
<b>Variable</b>	<b>Correlate</b>	<b>Correlation</b>	<b>p-value</b>
Fat Intake	Carbohydrate Intake	-0.873	<0.001
Protein Intake	Carbohydrate Intake	-0.767	<0.001
Fat Intake	Protein Intake	0.639	<0.001
Milk Consumption	Fat Intake	0.286	<0.001
Milk Consumption	Carbohydrate Intake	-0.280	<0.001
Fat Intake	Income	0.273	<0.001
Carbohydrate Intake	Income	-0.233	<0.001
Fat Intake	Sanitary Conditions	-0.252	<0.001
Milk Consumption	Father % Indian	-0.218	<0.001

\*Indian Blood Quantum was determined using Father % Indian and Mother % Indian and were thus highly correlated at 0.782 and 0.769, respectively.

eigenvalues greater than 1.5. The first factor is a loading reflecting dietary intake. The second is indicative of Indian blood quantum measures. The third factor loaded variables emphasizing environmental conditions.

The factor scores from these factors were used to construct the models tested below using a Partial Least Squares analysis (Table 7-18). Variables used as the dependent variables (Y) were TB, TB in the home, pneumonia, measles, heart problems, goiter, trachoma, other eye problems, rheumatism, and total illnesses in the home. Independent (X) variables were the factor scores from the diet, blood quantum, and environment factors. The model was a significant predictor (at  $p = 0.05$ ) of TB and its prevalence in households, goiters, trachoma, eye problems, and total illnesses per household. The coefficient of determination  $R^2$  was greatest for total illnesses per household.

Table 7-19 shows to what degree each individual factor in the model affected those variables identified as being significantly predicted by the model. The environment factor had

**TABLE 7-17. Factor analysis of variables from 1928. Positive loadings shown in green, negative loadings in yellow, and the highest communalities in blue.**

Variable	Diet	Blood Quantum	Environment	Communality
Income	0.275	0.419	0.454	0.457
Beds per Person	0.072	0.011	0.641	0.416
People in the Home	0.041	0.172	-0.756	0.603
Water Quality	-0.307	0.189	0.234	0.185
Sanitary Conditions	0.249	0.316	0.595	0.516
Pit Dug	-0.033	0.326	-0.112	0.120
Flies	-0.022	-0.056	0.457	0.212
Milk	0.490	0.305	-0.105	0.345
Carbohydrate	-0.960	0.012	-0.166	0.949
Fat	0.906	0.086	0.193	0.866
Protein	0.827	-0.163	0.086	0.718
Blood Quantum	-0.015	-0.933	-0.063	0.875
Father Quantum	-0.156	-0.765	-0.095	0.618
Mother Quantum	0.165	-0.802	-0.033	0.671
Variance	2.9594	2.6758	1.9161	7.5513
% Var	0.211	0.191	0.137	0.539

**TABLE 7-18. Partial least squares analysis with ANOVA of specific variables, using factor scores from the diet, blood quantum, and environment factors.**

VARIABLE	DF	SS	Error SS	MS	R-Sq	F	P
TB, diagnosed	3	17.85	784.154	5.95	0.022	6.1	0.000
TB, in the home	3	73.06	561.939	24.35	0.115	34.6	0.000
Pneumonia	3	6.31	795.697	2.10	0.008	2.1	0.097
Measles	3	7.70	794.311	2.57	0.010	2.6	0.052
Heart Problems	3	1.83	800.165	0.61	0.002	0.6	0.609
Goiter	3	29.95	772.055	9.98	0.037	10.3	0.000
Trachoma	3	12.47	789.526	4.16	0.016	4.2	0.006
Eye Problems	3	10.00	792.005	3.33	0.012	3.4	0.018
Rheumatism	3	4.31	797.693	1.44	0.005	1.4	0.230
Total Illnesses	3	318.34	483.661	106.11	0.397	175.3	0.000

**TABLE 7-19. Regression coefficients for variables significantly predicted by the PLS model.**

<b>FACTOR</b>	<b>TB, diagnosed</b>	<b>TB, in the home</b>	<b>Goiter</b>	<b>Trachoma</b>	<b>Eye Problems</b>	<b>Total Illnesses</b>
Diet Factor	0.0637	0.1890	0.0565	-0.1160	-0.0281	0.0678
Blood Factor	0.0063	-0.2309	-0.1471	-0.0195	-0.1024	-0.3326
Environment Factor	-0.1348	-0.1613	-0.1119	-0.0415	0.0347	-0.5308

illnesses per household. The coefficient of determination  $R^2$  was greatest for total illnesses per household.

Table 7-19 shows to what degree each individual factor in the model affected those variables identified as being significantly predicted by the model. The environment factor had the greatest impact on an individual having TB, while all three factors influenced TB in households, with the blood quantum factor being most influential. Goiters were influenced by the blood quantum and environment factors. Surprisingly, trachoma and other eye problems were not largely impacted by the environment factor. However, the environment factor had a dramatic influence on the total number of illnesses per household, with blood quantum also strongly influencing this variable.

## **DISCUSSION**

The Potawatomi Indian health survey ca. 1928 provided much needed information on the health and living conditions of the Prairie Band Potawatomi. Though these data were imperfectly recorded at a time when the value of data for analysis and evaluation were not fully recognized and the methodology not perfected, they do provide valuable information on the health and diet and living and economic conditions on the reservation.

Analyses of the 1928 data undertaken for this study suggest that income had a major impact on all aspects of Potawatomi life. Low income contributed to crowded living conditions,

poor water quality, poor sanitary conditions and an inadequate diet. These variables in turn increased the incidence of diseases per household and of some diseases in particular. Thus, being poor had a devastating impact on health and well-being. The PLS analysis indicates that the environment (of which income was a component) most strongly influenced the number of health problems experienced in households.

Having a greater degree of Indian ancestry generally guaranteed a lower income. The Dawes Act had placed the Potawatomi in a position to give up their land without adequate compensation, and Potawatomi land holdings had shrunk. Furthermore, the Potawatomi did not enthusiastically engage in commercial farming, imposed on them by the federal government. As a result, a group of people who already were economically and politically marginalized were further victimized as they were forced to adopt a subsistence strategy about which they knew and cared little (Clifton 1998). The average per capita income in 1929 was \$750 a year for all Americans; \$528 for Kansans; and the average annual income for someone working in agriculture was only \$273 (BEA 2006; McElvaine 1984). It is unlikely that many Potawatomi, including those characterized as having "good" incomes, made more than this average income for agriculturalists. The 1928 surveyors, in their summary, concluded that more than half of the Potawatomi they visited were indeed "poor."

The KSBH surveyors investigated the water quality on the reservation in 1928 because contaminated water contributes significantly to many diseases, including typhoid fever, cholera, and dysentery. Flies also spread these diseases as well as trachoma (USPHS 1913), and as early as 1914, Indian Commissioner Sells had instructed superintendents to render all reservation privies fly-proof (Putney 1980). The 1928 surveyors recorded whether the toilet facilities were protected from flies. Despite that the surveyors judged most of the water sources to be poor and

that most outhouses were not protected from flies, only a few households had experienced typhoid fever in the last five years, and no cases of typhoid or cholera were reported. Six cases of diarrhea were reported to the surveyors, and water and toilet facilities may have contributed to these illnesses among the Potawatomi. However, the low incidence of diseases caused principally by water and waste contamination suggests that drinking water and unsanitary toilets were not major contributors to disease on the reservation. Flies may have contributed to the high incidence of tuberculosis and trachoma among the Potawatomi, but other fly-borne diseases were uncommon. In this case, tuberculosis and trachoma probably were being spread by other means.

The Potawatomi in the late 1920s were not an urban population, but living conditions in many of the households were crowded. The mean number of people per household was greater than both the mean numbers of rooms and beds per house. Also, the KSBH surveyors described the sanitary conditions of almost half of the homes as being poor. The income of most Potawatomi was also well below that of most Americans. Thus, conditions on the reservation were ripe for tuberculosis.

Among the Potawatomi, however, tuberculosis was an equal opportunity infectious disease, and its prevalence did not vary among households that differed with regard to sanitary conditions or income. A possible explanation may be that the "variation" in sanitation and income was not great enough to confer protection for some or to increase risk for others. Tuberculosis was seen more often in homes that had more people. The likely explanation is that a larger household meant a larger pool of hosts. Also, poor public health infrastructure on the reservation may have contributed to the high prevalence of tuberculosis across socioeconomic groups. Health services were utilized by only half of the Potawatomi households, and most reported using these services only when absolutely necessary, long after the infections had

become entrenched. Nor is there any evidence that any kind of concentrated public health effort was undertaken on the Potawatomi Reservation.

Correlation analysis showed a relationship between measles and pneumonia. No other relationship between these diseases and the other variables was significant. The likely cause of the relationship between these two diseases is the role pneumonia plays as a secondary infection following a bout with measles and other viral diseases such as influenza. A primary infection of measles may cause an individual's lungs to be vulnerable to a pneumococcus bacilli infection (Keller 2002). Thus, households that suffered measles infections may have been more susceptible to pneumonia.

A variety of eye complaints (blindness, eye problems, sore eyes, poor eyesight, and trachoma) were the most often reported health problem among the Potawatomi in 1928. The only definitive diagnosis given was for trachoma; the remaining complaints came from patients rather than a health professional's diagnosis. "Sore eyes" is a phrase most often interpreted as trachoma, and the term was used often in the early part of the 20<sup>th</sup> century; trachoma may have been the principal cause of eye problems. Why the surveyors were unable to provide this diagnosis is unclear, though it is an additional indication that those conducting the survey were neither doctors nor nurses and were completely unfamiliar with the symptoms of the common maladies of the time and of the population. Likewise it is possible that "eye problems" referred to symptoms of conjunctivitis. This inflammation of the conjunctiva can be caused by viral and bacterial infections, as well as by chemical contamination or physical intrusions by dust or debris (Richards and Guzman-Cottrill 2010). Other potential eye disorders recognized at the time include chalazion, entropion (likely the result of trachoma), and pinguecula (a conjunctival degeneration). The lack of correlation between the incidence of trachoma and other eye



disorders and most other variables may be due to eye problems being so prevalent among the Potawatomi that variability in wealth or living conditions was insufficient to protect people from the disorders. Surveyors did inquire as to the use of individual towels among people and probably did so due to the already documented high incidence of trachoma on Indian reservations and Indian boarding schools. Only 4% of households that answered the question had individual towels for each family member, a lack of variability that may explain both the high prevalence of eye pathologies and the absence of correlation between eye problems and other variables.

Among the Potawatomi of 1928, diabetes was mentioned only twice, as the cause of death of a family member. No one living on the reservation at the time was diagnosed with diabetes, but at the time this would have involved an examination of the urine, and there is no indication that the surveyors ever went to great lengths to diagnose any disease or were properly trained to do so. With poor access to professional medical services, more Potawatomi may have suffered from the disease than were ever diagnosed with it. In fact, the high rate of eye complaints among the Potawatomi may be an early indicator that diabetes was becoming prominent. Unfortunately, the KSBH surveyors also neglected to record height and weight, which would have been useful indicators that people suffering from eye disorders might also be diabetic.

Enlarged thyroids, or goiters, were common among the Potawatomi. The condition is due primarily to a lack of iodine in the diet (Maberly 1994). Risk factors include female sex, age over 40 years, residence in an endemic area, ingestion of large amounts of goitrogenic foods, and a family history of goiter. Regions where iodine concentrations in the soil are low produce crops and wild plants that have low levels of iodide, contributing to iodine deficiency in their

consumers. The geographical areas with iodine-depleted soil are usually montane or at some distance from oceans, which provide iodine *via* the iodine cycle. The Great Lakes, Intermountain regions, and the Midwest are the most iodine-deficient regions in the U.S. Among the Potawatomi reported to have enlarged thyroid glands, all of those whose sex could be ascertained were female. Goiters in general are more common in women and are associated with female sex hormones and iodine needs during pregnancy. In particular, thyroid-stimulating hormone (TSH) levels increase during pregnancy, and TSH and estrogen are associated with an increased risk of goiter (Farahati et al. 2006).

Goiters may also be caused by goitrogens, substances in foods that interfere with thyroid gland function. Goitrogenic foods include raw broccoli, brussel sprouts, cabbage, cauliflower, cassava, rutabagas, and turnips (Greer 1957). Cabbage was one of the few vegetables available to even the poorest Potawatomi. Cooking destroys the enzymes involved in the formation of goitrogenic substances in plants, however, and generally large amounts of goitrogenic foods must be consumed before thyroid gland function is impaired. Thus, goitrogenic foods are a less probable cause of goiters among the Potawatomi than a simple lack of iodine in the foods they ate. The low iodine content of the soil of Kansas resulted in crops and wild plants that were deficient in iodine, leading to goiters among a number of Potawatomi women. Today, consumption of iodized salt is the primary method for preventing iodine-deficiency disease, but iodized salt was probably not available to the Potawatomi at the time of the survey. Research on the efficacy of adding iodine to salt for preventing goiters had begun only recently (see Kimball 1928a, 1928b and Olin 1924) and was focused primarily in the Great Lakes region.

Food recommendations of the time were based on USDA food guides originally developed in 1916 and modified slightly in 1917, 1921, and 1923. These guides categorized

foods according to five different groups: meats and milk, cereals, fruits and vegetables, fatty foods, and sugary foods. Recommendations were that meats (10%) and milk (10%) together constitute 20% of the total diet; cereals, consisting of grains and other starchy foods such as potatoes, 20%; fruits and vegetables, 30%; fatty foods, 20%; and sugary foods, 10% (Davis and Saltos 1999). Modern dietary recommendations still emphasize cereals (~36%), followed by “protein foods” (29%), vegetables (~13%), dairy (~13%), and fruits (~9%). Sweets and fats are to be used only sparingly. The shift since the 1920s has obviously been to emphasize cereal intake and minimize fat and sugar intake. More current recommendations are that for adults, 45-65% of dietary calories should come from carbohydrate, 20-35% from fat, and 10-35% from protein (USDA and DHHS 2010).

The Potawatomi diet on average was 46% carbohydrate, 27% fat, and 27% protein. This is a relatively high protein diet by comparison with the recommendations of the day. The traditional diet of many Native American populations was high in protein due to their reliance on hunting wild game (Price 2003): precontact Potawatomi subsistence consisted of maize horticulture combined with hunting, fishing, and gathering. Even as corn agriculture became more complex and productive, meat was still the primary source of protein and was additionally valued for ritualistic purposes (Clifton 1998). The relationship between the degree of “Indian blood” of mothers and protein intake may reflect a Potawatomi woman's cultural food preferences and the tendency for more traditional Potawatomi families to rely on wild game. Meat was not the only major source of protein for the Potawatomi in the 1928 survey, however. Beans, another traditional Indian food that is high in protein, were widely consumed as well.

Intake of wild game must be considered as having been substantial, given the reports of some older Potawatomi who grew up on the reservation and are living today. They have stated

that hunting was a frequent source of food when they were young. Hunted animals included deer, rabbits, squirrels, raccoons, quail, pheasant, prairie chickens, fish, frogs, turtles, muskrats, and skunks. These sources of meat seem to have been overlooked by the surveyors, probably because they did not fit into the Euro-American development schemes being pushed upon the Potawatomi.

Also inadequately recorded by the surveyors was the variety of wild plants utilized by the Potawatomi, whereas wild plants named by Potawatomi elders who were living in 1928 include milkweed, wild onions, nettles, strawberries, plums, gooseberries, grapes, chokecherries, Indian potatoes, papaws, blackberries, raspberries, persimmons, dewberries, black walnuts, acorns, hazelnuts, and hickory nuts (Figure 7-4). From this list of foods, only "wild berries," gooseberries, and grapes were mentioned by the surveyors; and, reportedly, they were consumed by very few families. Neither did the surveyors mention that many Potawatomi annually grew, dried, and stored a maize crop of the traditional type (Clifton 1998). Traditionally grown and prepared maize undoubtedly enhanced energy and nutrient intake of some of the people. The surveyors did recognize the contribution of the summer gardens to dietary intake, probably because gardening conformed more to their view of appropriate subsistence than gathering wild plants or growing traditional foods.

The Prairie Band Potawatomi most certainly supplemented their diets with gathered and hunted foods, which were primarily ignored by the KSBH surveyors. Therefore, it is difficult to say that the government-supported agricultural schemes contributed significantly to a reduction in illness due to better nutrition. More than likely, the Potawatomi achieved a balanced diet



**FIGURE 7-4. Potawatomi woman with wild fruits, probably persimmons, Prairie Band Reservation, 1930s. (courtesy Denver Public Library, Western History Collection).**

because they continued to utilize traditional subsistence activities that increased their protein and micronutrient intake, but they also incorporated government rations into their diet, thus supplementing their total caloric intake. This may explain the lack of correlation between dietary intake and illnesses such as tuberculosis among the Potawatomi.

The surveyors reported that Indian children were particularly fond of milk but its expense was a barrier. That milk consumption correlated negatively with the degree of Indian ancestry of fathers may reflect the fact that milk is not a traditional Indian food and thus was not consumed in households where the father was predominantly Indian. Due to this and its cost, fewer than

half of the households had milk available to them. What the surveyors did not know was that up to 80% of Native Americans are lactose intolerant (Flatz 1987). The less frequent consumption of milk in households where the father had a higher degree of Indian blood may reflect an avoidance of milk due to symptoms of lactose intolerance.

The inability to digest lactose, or milk sugars, after infancy is common throughout many of the world's populations. Without sufficient lactase enzyme activity, lactose cannot be digested. Symptoms of lactose intolerance include gas, bloating, abdominal discomfort, and diarrhea. The diarrhea may be severe enough to cause dehydration as well as the elimination of essential nutrients before they can be absorbed (Beers 2003; Beers and Berkow 1999). Milk consumption by Indian children and adults may have compromised their health and made them more vulnerable to infections (Keller 2002). Among the Potawatomi in 1928, milk consumption had no effect on the incidence of tuberculosis, but lactose intolerance resulting in severe diarrhea may have interfered with absorption of nutrients important for eye health, such as vitamins A, C, and E. Diarrhea was not reported to be a major condition among the Potawatomi in 1928 but Potawatomi elders do recall it as a health problem, particularly among children. Perhaps it was so common that it was simply overlooked. Whether or not lactose intolerance was a primary cause of the cases of diarrhea is not known, but unlikely given the low levels of milk consumption.

## **Conclusions**

Though it was ostensibly an assessment of health, the 1928 survey was as much about assimilation as it was health. In their summary, the surveyors criticized the Potawatomi for not embracing the agricultural economy forced upon them. They blamed poverty primarily on the Potawatomi themselves. Criticism was leveled at the management skills of Potawatomi men as

farmers. Their wives were criticized for their lack of industry in domestic affairs. Children were described as "incorrigible," and parents were blamed for the lack of discipline of their offspring. Potawatomi adolescents were criticized for their immorality and irresponsibility. All of this criticism exposed the surveyors' deeply ethnocentric and implicitly racist views.

The diseases affecting the Potawatomi at the time of the 1928 survey were primarily infectious. Tuberculosis, pneumonia, measles, influenza, trachoma, typhoid, and smallpox were reported. The prevalence of these conditions indicates that the Potawatomi in 1928 had not yet begun the epidemiological transition that much of the U.S. population was undergoing. The National Center for Health Statistics had reported diseases of the heart as the fourth most common cause of death in the U.S. population in 1900, and as the number one cause of death by 1910 (Linder and Grove 1947). Between 1900 and 1928, tuberculosis went from the first to the sixth-ranked cause of death, but among the Prairie Band Potawatomi tuberculosis still was the most common killer in 1926 and 1927 and the most commonly reported cause of death on the 1928 survey. Nor did the Prairie Band Potawatomi resemble non-Indian residents of Kansas with regard to disease prevalence. In 1928 in the state of Kansas (Table 7-20), diseases of the heart were the number one cause of death, whereas tuberculosis was the seventh leading cause (and less prevalent than in the rest of the United States) (see Table 7-7 for the leading causes of death among the Potawatomi). Morbidity and mortality among the Potawatomi in 1929 resembled that of most of America in the 19<sup>th</sup> century, reflecting the absence of improvements in sanitation, a cleaner water supply, and public health programs on the reservation. These data and information are consistent with a population yet to complete the transition into the *Age of Degenerative and Man-Made Diseases*.

**TABLE 7-20. Top 15 causes of death in Kansas, 1928 (Bureau of the Census 1930:350).**

<b>Cause</b>	<b>Number</b>	<b>Percent</b>
Diseases of the heart	2972	14.19
Cerebral hemorrhage	2102	10.04
Nephritis	1632	7.79
Influenza	1472	7.03
Other external causes	1061	5.07
Pneumonia	1254	5.99
Tuberculosis of the respiratory system	644	3.07
Diabetes mellitus	376	1.80
Automobile accidents	364	1.74
Diarrhea and enteritis [under 2 years]	309	1.48
Suicide	243	1.16
Other puerperal causes	158	0.75
Diarrhea and enteritis [2 years and over]	121	0.58
Bronchitis	113	0.54
Puerperal septicemia	103	0.49

## **Chapter 7 References Cited**

BEA (Bureau of Economic Analysis). 2006. SA 1-3 March 2006 / Revised September 2006. U.S Department of Commerce.

Beers, M.H., ed. 2005. "The Merck Manual of Medical Information," Second Home Edition, Online Version. Whitehouse Station, NJ: Merck Research Laboratories. May 16, 2005.  
<http://www.merck.com/mmhe/sec09/ch129/ch129c.html?qt=diarrhea&alt=sh#sec09-ch129-ch129c-712>

Beers, M.H. and Berkow, R., eds. 1999. *The Merck Manual of Diagnosis and Therapy*. Whitehouse Station, NJ: Merck Research Laboratories.

Bureau of the Census. 1930. *Mortality Statistics 1928*. Department of Commerce. Washington, DC: U.S. Government Printing Office.

Clifton, James, A. 1998. *The Prairie People: Continuity and Change in Potawatomi Indian Culture, 1665 - 1965*. Iowa City: University of Iowa Press,

Davis, C. and Saltos, E. 1999. "Dietary Recommendations and How They Have Changed Over Time," in *America's Eating Habits: Changes and Consequences*. Edited by E. Frazao, pp. 33-50. Agriculture Information Bulletin No. 750.

Farahati, J., Wegscheider, K., Christ, K., Gilma, E., and Oing, W. 2006. Gender-Specific Determinants of Goiter. *Biological Trace Element Research* 113:223-230.

Flatz, G. 1987. "Genetics of Lactose Digestion in Humans," in *Advances in Human Genetics*. Edited by H. Harris and K. Hirschhorn, pp. 1-77. New York: Plenum Press.



- Greer, M.A. 1957. Goitrogenic substances in food. *American Journal of Clinical Nutrition* 5(4):440-4.
- Hill, T and Lewicki, P. 2006. *Statistics: Methods and Applications*. Tulsa, OK: Statsoft, Inc.
- Kaiser, H. F. 1960. The application of electronic computers to factor analysis. *Educational and Psychological Measurement* 20: 141-51.
- Keller, J.A. 2002. *Empty Beds: Indian Student Health at Sherman Institute, 1902 - 1922*. East Lansing: Michigan State University Press.
- Kimball, O.P. 1928a. Endemic goiter and public health. *American Journal of Public Health* 18:587-601.
- . 1928b. The efficiency and safety of the prevention of goiter. *JAMA* 91:454-460.
- Linder, F.E. and Grove, R.D. 1947. *Vital Statistics Rates in the United States 1900 – 1940*. National Office of Vital Statistics. Washington, DC: U.S. Government Printing Office.
- Maberly, G.F. 1994. Iodine Deficiency Disorders. *Journal of Nutrition* 124:1473S - 1478S.
- McElvaine, Robert S. 1984. *The Great Depression*. New York: Times Books.
- Olin R.M. 1924. Iodine deficiency and prevalence of simple goiter in Michigan. *JAMA* 82:1328-1332.
- Price, W.A. 2003. *Nutrition and Physical Degeneration*, 6<sup>th</sup> edition. La Mesa, CA: Price-Pottenger Nutrition Foundation.
- Putney, Diane T. 1980. *Fighting the Scourge: American Indian Morbidity and Federal Policy, 1897 – 1928*. PhD Dissertation. Milwaukee, WI: Marquette University.
- Richards, Amanda and Judith Guzman-Cottrill, J. 2010. Conjunctivitis. *Pediatrics in Review* 31:196-208.
- USDA (U.S. Department of Agriculture) and DHHS (Department of Health and Human Services). 2010. *Dietary Guidelines for Americans 2010*, 7th Edition. Washington, DC: U.S. Government Printing Office.
- USPHS (U.S. Public Health Service). 1913. *The Prevalence of Contagious and Infectious Diseases among the Indians of the United States*. Senate Document No. 1038, 62<sup>nd</sup> Congress, 3<sup>rd</sup> session, serial 6365.

## **Chapter 8: Mortality 1974 – 2004**

### **Introduction**

As the 20<sup>th</sup> century came to a close, chronic conditions such as diabetes were killing more and more Native Americans. The dramatic increase and extreme levels for some diseases, particularly metabolic conditions, became a concern for public health officials. The purpose of this analysis is to quantify the deaths among Native Americans in Jackson and Shawnee counties, which encompass a large number of Prairie Band Potawatomi tribal members, as well as members of other tribes such as the Kickapoo. Of particular interest are the contributing factors to the observed pattern over the 1974-2004 time period. Quantifying mortality of the population also allows for comparison with data from earlier in the century, providing the opportunity to examine the epidemiologic transition in this area over a longer time.

### **Methods**

#### **Data**

Data on cause of death are available through the Office of Vital Statistics at the Kansas Department of Health and Environment (KDHE). The data also include age and year of death, county, and sex of the decedent, for the years 1974–2004. Unfortunately, prior to 1974 the KDHE did not identify “Native American” or “American Indian” as a separate racial category, so statistics on Indian deaths in Kansas prior to 1974 are difficult to locate, if not completely nonexistent. The records do not include information on tribal affinity, so all records for Native Americans from the counties Jackson and Shawnee counties were used. These records include Prairie Band Potawatomi but also include people from other tribes and are indicated below as the JK/SH community (for a more detailed discussion of the “Prairie Band Community” see Appendix B). The KDHE Office of Vital Statistics uses the International Statistical

Classification of Diseases and Related Health Problems (ICD) codes to record deaths. Causes of death were recorded using ICD-9 codes for 1974–1998 and ICD-10 codes for 1999–2004.

Records of the State of Kansas Library provided the total Native American population data for the state, by county and by age groups for 1970 and from 1980 to 2004. These data were used to construct age-adjusted death rates using the standard population weights published by the CDC, which represent the proportion of the U.S. 2000 population by each age group. State of Kansas Library data were combined with the KDHE data to produce age-adjusted death rates.

### **Statistical methods**

Frequencies were constructed from these data to show changes in mortality over time. Regression trend lines were fitted to the observed values. The primary purpose of this was to model a smooth trajectory of deaths from the highly variable data resulting from low sample numbers, providing a visual representation of mortality trends by cause. Linear regression lines were modeled on the data to provide an estimated up or down tendency. As expected, the fits ( $R^2$  values) of the models were generally poor due to the extreme variability of the data.

According to the National Center for Health Statistics, death rates for the Native American population must be interpreted with caution due to reporting problems related to incorrect identification of race on both death certificates and in population censuses and surveys. As a result of reporting problems, Native American mortality rates may be underestimated by as much as 30% (Arias et al. 2008; Heron et al. 2009). Annual death rates based on fewer than 20 cases are considered to be unstable due to a large relative standard error (RSE), the standard error divided by the mean and expressed as a percentage. Modern mortality statistics for Native Americans are also problematic from this perspective. To manage the small number of annual deaths from some causes, multiple years were combined to produce an average mortality more

representative of the region and comparable to standard populations. The specific years combined are identified in each instance. To determine if age-adjusted rates were significantly different from the standard population, a standardized mortality ratio (SMR) was computed (see Table 8-5):

$$\text{SMR} = \text{observed/expected (from the standard population)}$$

The 95% confidence intervals were computed as:

$$1.96 \times \text{seSMR}$$

with the standard error of SMR being:

$$\text{seSMR} = \sqrt{\text{observed/expected}}$$

such that the upper limit equals:

$$\text{SMR} + 1.96 \times \text{seSMR}$$

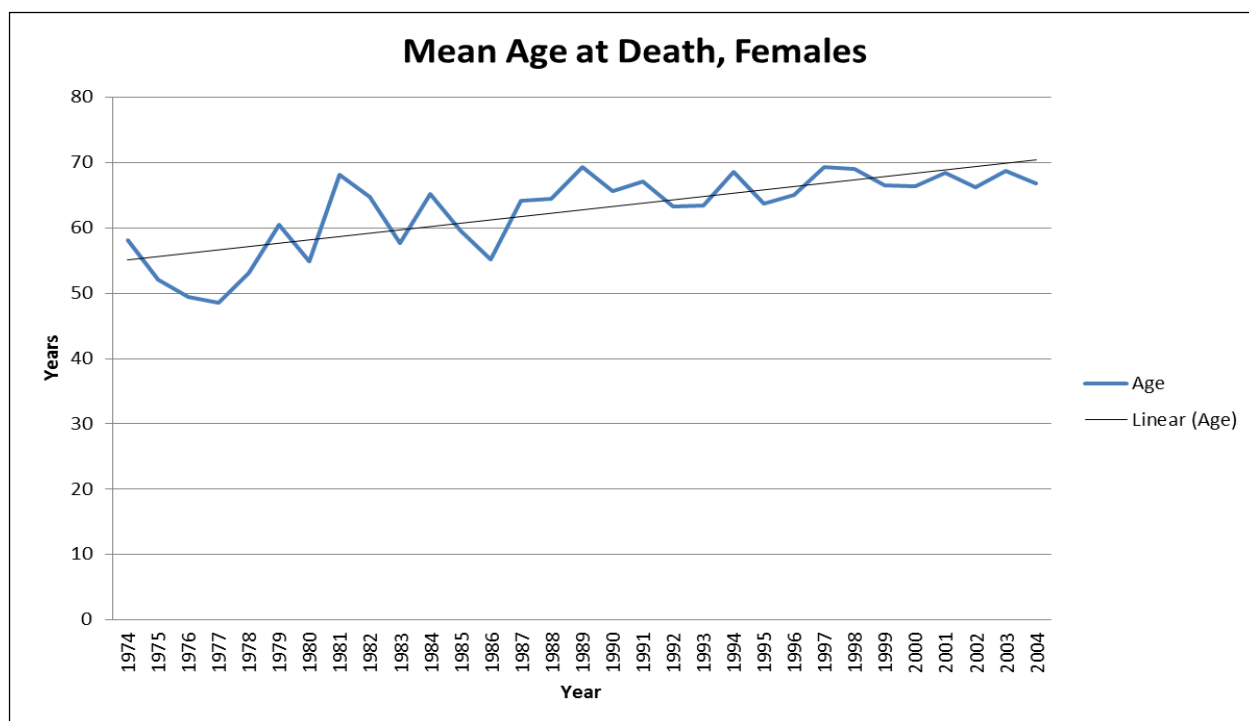
and the lower limit equals:

$$\text{SMR} - 1.96 \times \text{seSMR}$$

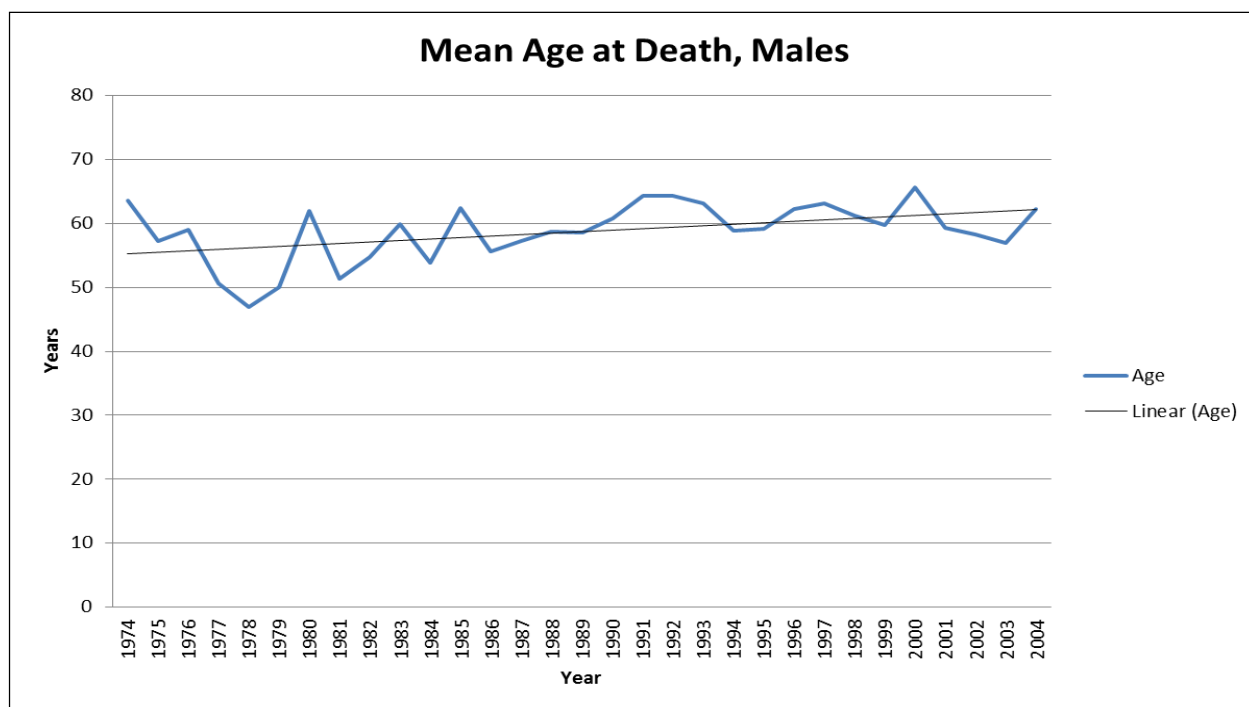
Values were considered significantly different when the 95% confidence interval did not include 1 (Curtin and Klein 1995).

## Results

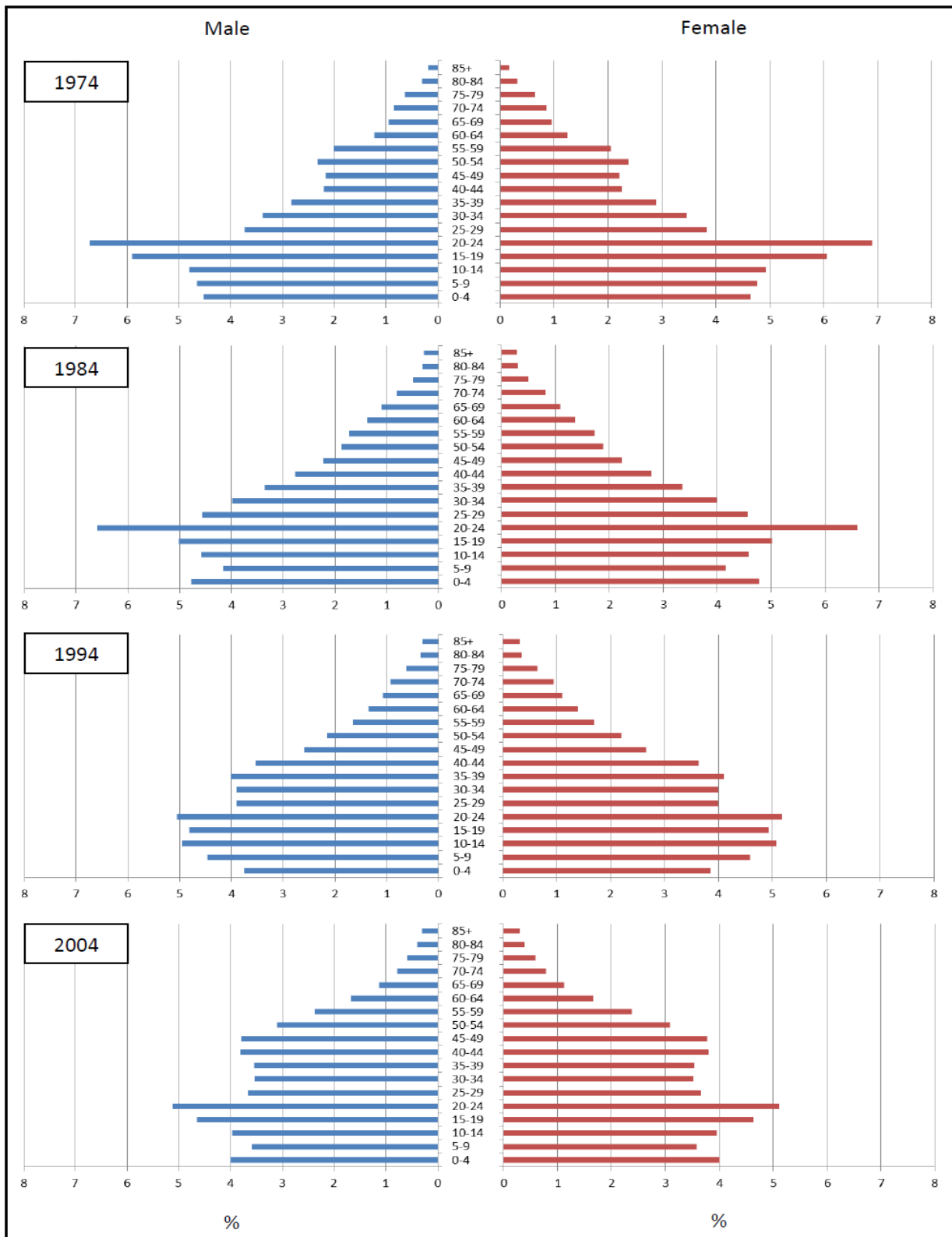
Native Americans of the region were living longer over time. Mean age at death increased between 1974 and 2004, from 61.5 years to 64.4, with a low of 49.9 years in 1977 and a high of 66.1 years in 2000. Females showed a slightly greater increase in age at death (Figure 8-1) over time than males (Figure 8-2).



**FIGURE 8-1. Mean age at death among females in Jackson and Shawnee counties, 1974-2004, with linear trend line.**



**FIGURE 8-2. Mean age at death among males in Jackson and Shawnee counties, 1974-2004, with linear trend line.**



**FIGURE 8-3. Population pyramids showing the age distributions of Native Americans in Jackson and Shawnee counties, 1974, 1984, 1994, and 2004.**

An analysis of the age distribution of the population between 1974 and 2004 indicated a younger population in 1974 than in 2004 (Figure 8-3), showing the highest peaks for younger ages (20-24) in the first two decades, and lower percentages for the youngest age group (0-4) in the latter two decades. The 2004 data produced a pyramid more similar to those seen among developed nations. For chronic diseases, age-adjusting the crude rates tended to increase the most dramatic peaks, further indicating the generally younger population of Native Americans in Jackson and Shawnee counties compared to the standard population.

By 1974 heart disease had become the most common cause of death among Native Americans in Jackson and Shawnee counties, followed by cancers, stroke, accidents, and diabetes (Table 8-1), indicating that the population had already passed into the Age of

**TABLE 8-1. Age-adjusted causes of death by percent among the Native Americans in Jackson and Shawnee counties 1974, 1980, 1985, 1990, 1995, 2000, 2004, and ranked by mortality rate, 1974-2004.**

Condition	1974	1980	1985	1990	1995	2000	2004	Rank
Heart Disease	33.3	23.5	42.9	66.7	20.0	28.6	18.2	1
Cancers	5.6	11.8	0.0	11.1	20.0	14.3	18.2	2
Stroke	5.6	23.5	7.1	0.0	13.3	9.5	9.1	3
Accidents	5.6	0.0	21.4	0.0	6.7	4.8	18.2	4
Diabetes	0.0	0.0	14.3	11.1	6.7	4.8	4.5	5
Other	0.0	5.9	0.0	0.0	13.3	9.5	9.1	6
Influenza and Pneumonia	16.7	0.0	0.0	0.0	6.7	9.5	0.0	7
Homicide	11.1	11.8	0.0	0.0	0.0	0.0	0.0	8
Atherosclerosis	5.6	0.0	7.1	0.0	0.0	4.8	0.0	9
Suicide	0.0	5.9	0.0	11.1	0.0	0.0	0.0	10
Other diseases of the circulatory system	5.6	5.9	0.0	0.0	0.0	0.0	0.0	11
Other diseases of the respiratory system	0.0	5.9	0.0	0.0	0.0	4.8	0.0	12
Perinatal Conditions	5.6	0.0	0.0	0.0	0.0	4.8	0.0	13
Other diseases of the digestive system	5.6	0.0	0.0	0.0	0.0	0.0	4.5	14
Kidney Failure	0.0	0.0	0.0	0.0	0.0	0.0	9.1	15
Other diseases of the nervous system	0.0	0.0	7.1	0.0	0.0	0.0	0.0	16
Hypertensive heart and renal disease	0.0	0.0	0.0	0.0	6.7	0.0	0.0	17
Liver Diseases	0.0	0.0	0.0	0.0	6.7	0.0	0.0	17
Other infectious and parasitic diseases	0.0	5.9	0.0	0.0	0.0	0.0	0.0	19
Other endocrine, nutritional and metabolic diseases	0.0	0.0	0.0	0.0	0.0	4.8	0.0	20
Alzheimer's disease	0.0	0.0	0.0	0.0	0.0	0.0	4.5	21
COPD	0.0	0.0	0.0	0.0	0.0	0.0	4.5	21
Emphysema	0.0	0.0	0.0	0.0	0.0	0.0	0.0	23
Other diseases of the genitourinary system	0.0	0.0	0.0	0.0	0.0	0.0	0.0	23
Septicemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	23
SIDS	0.0	0.0	0.0	0.0	0.0	0.0	0.0	23
Tuberculosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	23

**TABLE 8-2. Comparison of change in death rates from 1974 – 2004 for Native Americans in Jackson and Shawnee counties and for all Kansas Native Americans. Numbers are based on linear trend lines on age-adjusted death rates per 100,000 population.**

Cause	Change JK/SH	Change KS
Cancer	118	110.0
COPD	35	32.0
Liver Disease	32	2.0
Septicemia	15	5.0
Accidents	1	3.5
Suicide	-2	6.5
Kidney Failure	-4	9.5
Heart Disease	-5	67.0
Diabetes	-9	17.5
Influenza and Pneumonia	-20	-3.0
Stroke	-50	14.0
Homicide	-55	-14.0

Degenerative and Man-Made Diseases. Influenza and pneumonia demonstrated their highest percentage of deaths in 1974, with lower levels in subsequent years. The yearly variation during the period most likely represents the small sample sizes of deaths for each year.

According to these data, among the Native Americans in Jackson and Shawnee counties, cancers had the most dramatic increase as causes of death between 1974 and 2004 (Table 8-2). Chronic obstructive pulmonary disease (COPD), liver disease, septicemia, and accidents also showed increases during this time period. Suicide, kidney failure, heart disease, and diabetes showed slight decreases, whereas homicides, strokes, and influenza and pneumonia showed more dramatic decreases. The age-adjusted death rate among Native Americans in Jackson and Shawnee counties was essentially flat between 1974 and 2004 (Figure 8-4).

Death rates from the small Jackson and Shawnee county samples were compared to the death rates for all Native Americans in Kansas (Table 8-2). Increases in cancer and COPD were consistent with the Native American data statewide; whereas heart disease, diabetes, stroke,



kidney failure, suicide, septicemia, accidents, and liver disease increased among Native Americans statewide and decreased in the smaller sample. Only homicides and influenza and pneumonia showed decreases among all Kansas Native Americans.

To get a better idea of recent mortality rates in the tribe, an average was computed by each cause across the years 1990–2004 (Table 8-3). Heart disease was number one, followed

**TABLE 8-3. “Recent” age-adjusted causes of death by percent among Native Americans in Jackson and Shawnee counties, 1990-2004.**

Condition	1990-2004 AVG	1990-2004 RANK
Heart Disease	30.3	1
Cancers	19.7	2
Accidents	8.7	3
Stroke	5.2	4
Diabetes	4.4	5
Liver Diseases	4.3	6
Other diseases of the digestive system	3.3	7
Influenza and Pneumonia	2.6	8
COPD	2.6	9
Kidney Failure	1.8	10
Other diseases of the respiratory system	1.4	11
Other endocrine, nutritional and metabolic diseases	1.2	12
Alzheimer's disease	1.2	12
Suicide	1.0	14
Other diseases of the nervous system	0.9	15
Homicide	0.8	16
Perinatal Conditions	0.8	16
Septicemia	0.8	16
Other diseases of the circulatory system	0.7	19
Tuberculosis	0.4	20
Other infectious and parasitic diseases	0.4	20
Hypertensive heart and renal disease	0.4	20
Atherosclerosis	0.3	23
Emphysema	0.3	23
Other diseases of the genitourinary system	0.2	25
SIDS	0.0	26

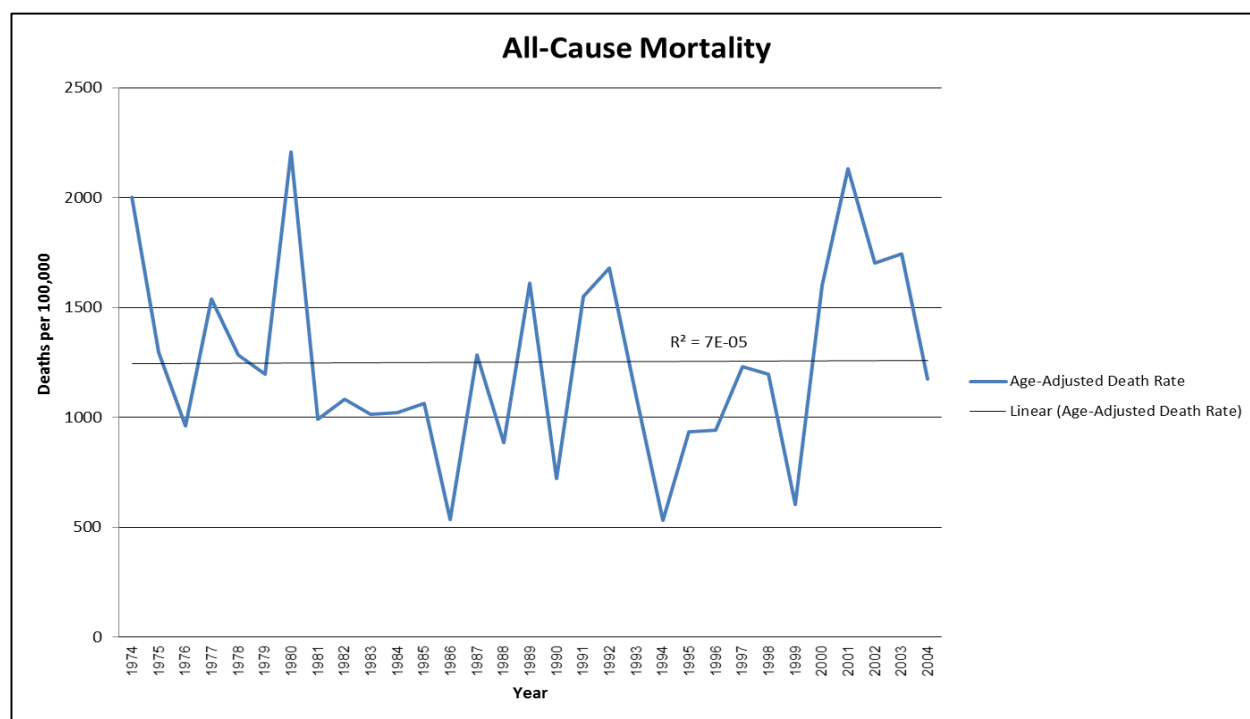
by cancers, accidents, strokes, and diabetes, respectively. The increasing trend in cancer mortality, the second most common cause of death after 1974, was the most dramatic change in the principal causes of mortality among Native Americans in Jackson and Shawnee counties. Cancer deaths by type are presented in Table 8-4. Cancers of the lung were the most common after 1990, followed by cancers of the colon, stomach, and ovaries.

The age-adjusted death rate for Native Americans in Jackson and Shawnee counties produced a trend line showing declines in the 1980s with increases thereafter (Figure 8-4), corresponding to the onset of the obesity epidemic. Compared to the overall U.S. age-adjusted

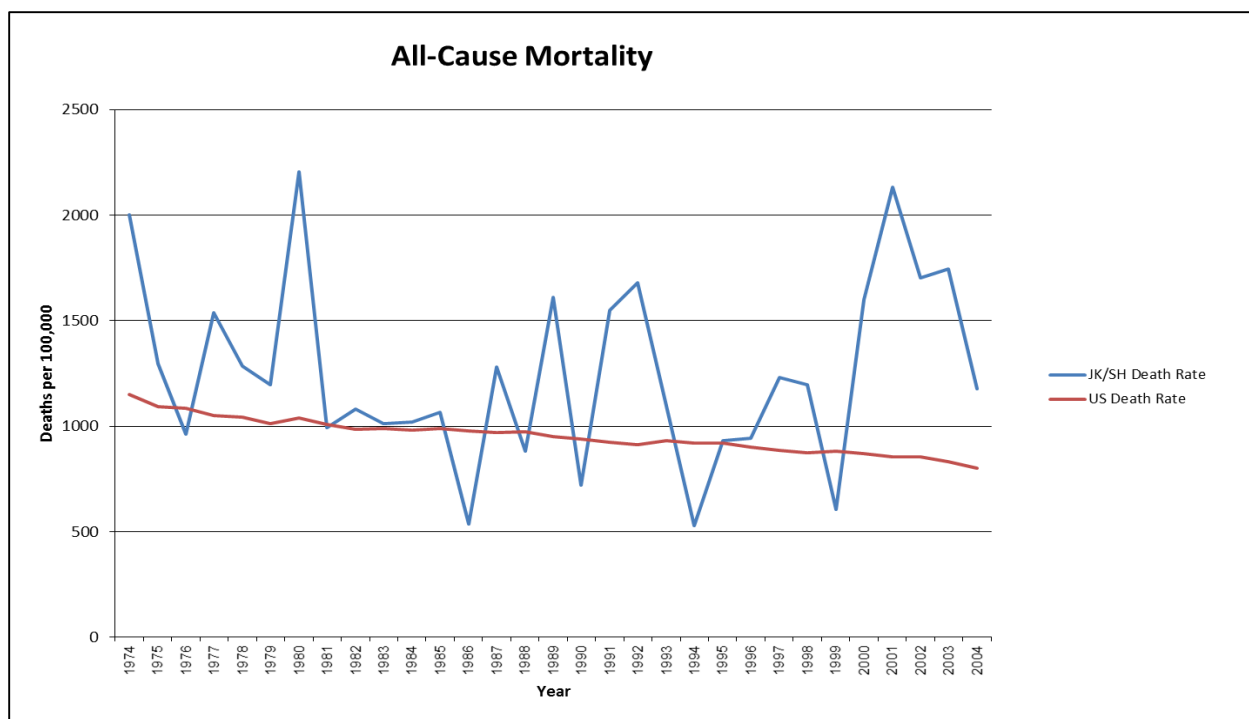
**TABLE 8-4. Cancer deaths by type, Native Americans in Jackson and Shawnee counties, 1990-2004.**

Cancer Type/Site	Percent of Total Cancers
Malignant neoplasm of trachea bronchus and lung	42.1
Malignant neoplasm of colon	12.3
Malignant neoplasm of stomach	8.8
Malignant neoplasm of ovary	5.3
Malignant neoplasm of kidney	3.5
Leukemia	1.8
Malignant neoplasm of anus	1.8
Malignant neoplasm of breast	1.8
Malignant neoplasm of esophagus	1.8
Malignant neoplasm of liver	1.8
Malignant neoplasm of major salivary glands	1.8
Malignant neoplasm of multiple myeloma	1.8
Malignant neoplasm of nasopharynx	1.8
Malignant neoplasm of pancreas	1.8
Malignant neoplasm of prostate	1.8
Malignant neoplasm of rectum	1.8
Malignant neoplasm of skin	1.8
Malignant neoplasm of tongue	1.8
Malignant neoplasm of urethra	1.8
Non-Hodgkin's lymphoma	1.8
Other and unspecified malignant neoplasms	1.8

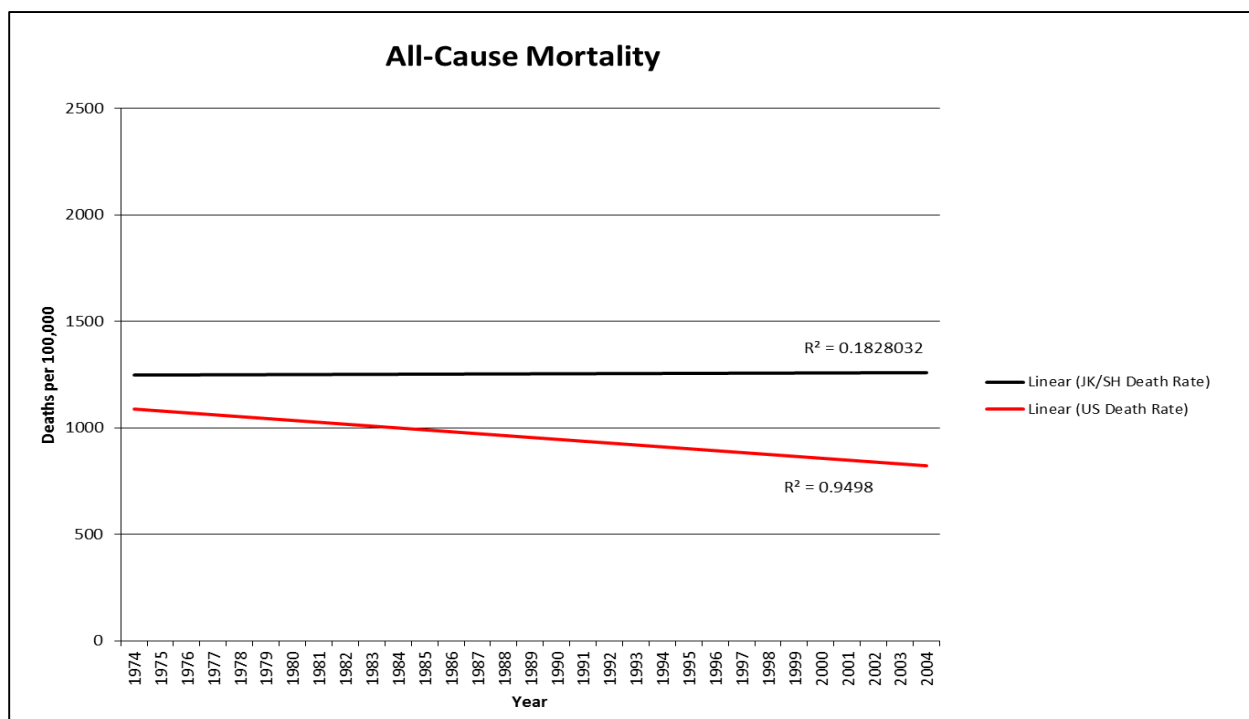
death rate (Figure 8-5), the death rate of Native Americans in Jackson and Shawnee counties did not decrease over the same time period (Figure 8-6). Graphs of specific causes of death, adjusted for age are presented below. The trends for heart disease (Figure 8-7), strokes (Figure 8-9), diabetes (Figure 8-11), influenza and pneumonia (Figure 8-13), and homicides (Figure 8-18) were downward, only slightly in most cases, but more dramatically for homicides. Diabetes deaths were highest in 1977 and most consistent during the years 1988-1995. Accidents (Figure 8-10) showed an upward trend over the 30-year time period, but with recent decreases from the peak years of the early 1990s. Suicides (Figure 8-17) were more common prior to 1991, suggesting a slightly downward trend. Kidney disease deaths (Figure 8-15) remained at approximately the same level from 1974 to 2004, but dipped in the years between. Cancers



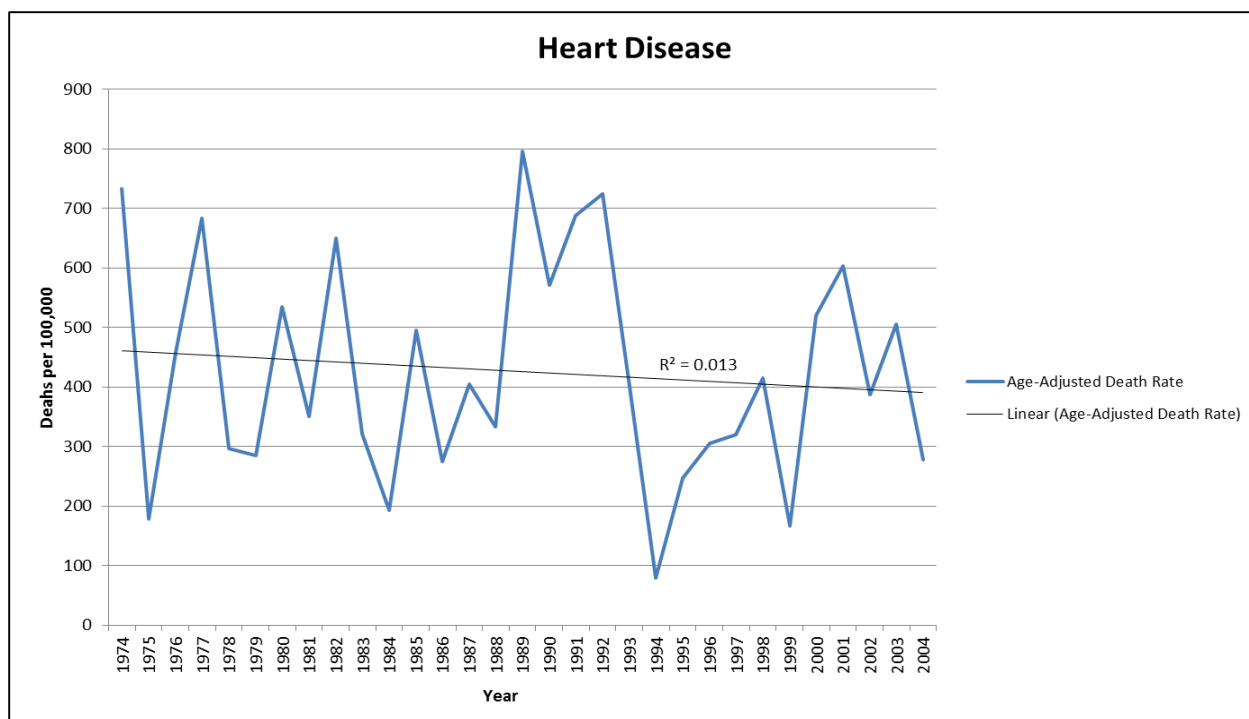
**FIGURE 8-4. Age adjusted death rates per 100,000 for all causes of mortality among Native Americans in Jackson and Shawnee Counties, with linear trend line.**



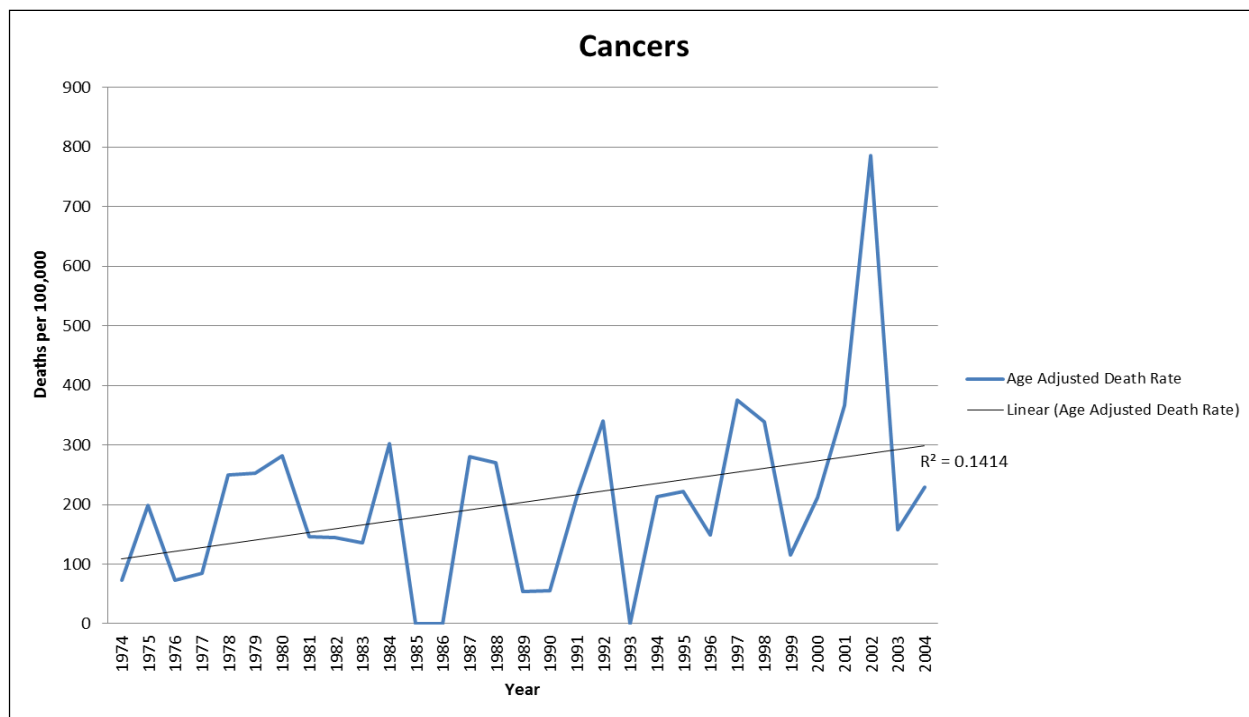
**FIGURE 8-5. Native Americans in Jackson and Shawnee counties vs. US age-adjusted death rates per 100,000 population.**



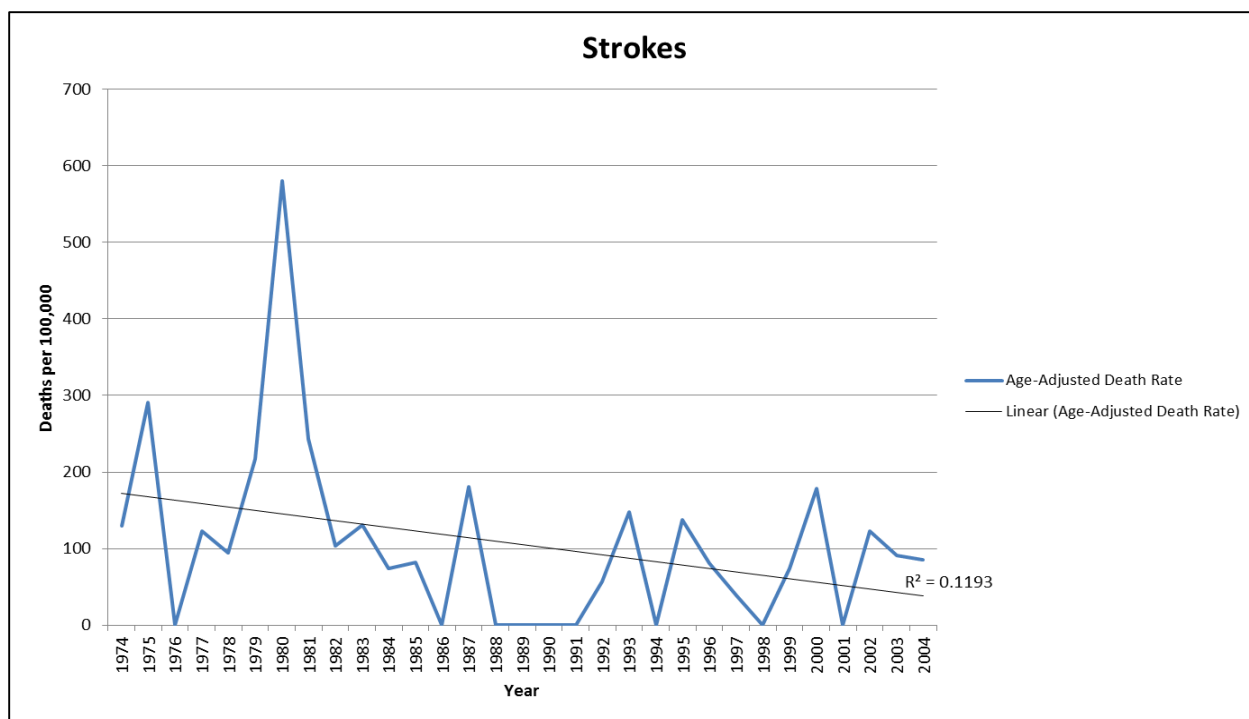
**FIGURE 8-6. Trend lines for Native Americans in Jackson and Shawnee counties vs. US age-adjusted death rates per 100,000 population.**



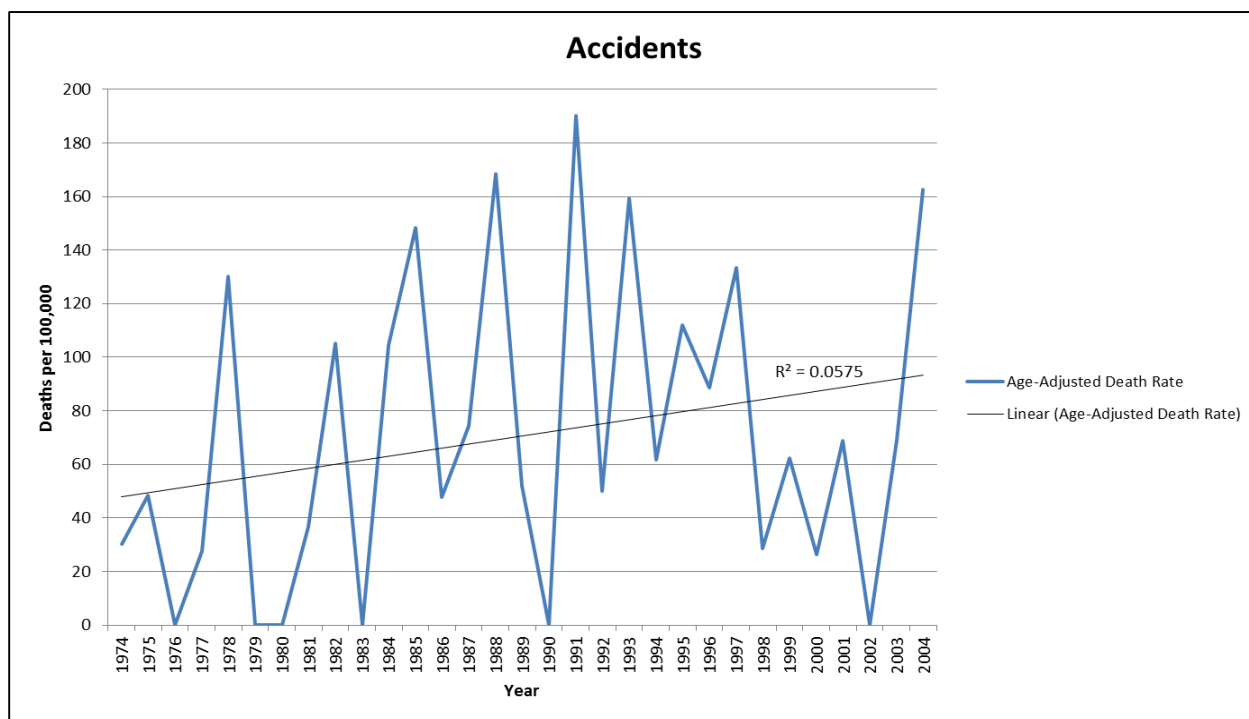
**FIGURE 8-7. Age adjusted death rates per 100,000 for heart disease among Native Americans in Jackson and Shawnee Counties, with linear trend line.**



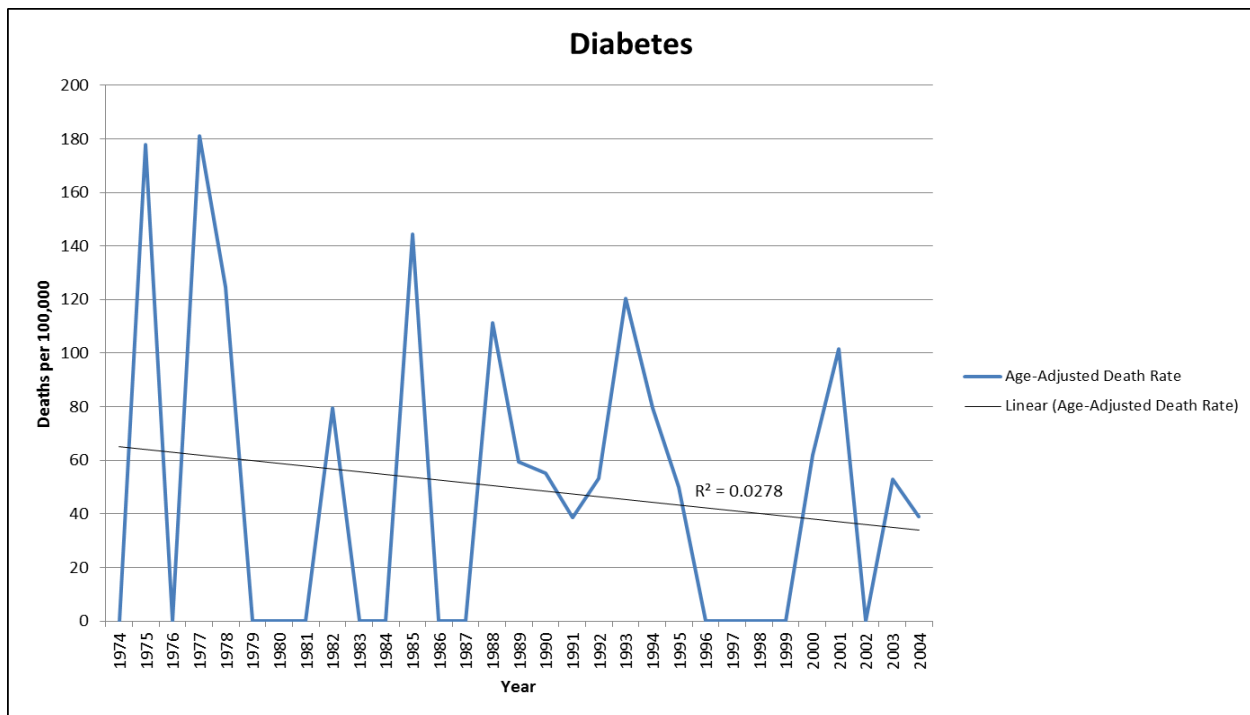
**FIGURE 8-8. Age adjusted death rates per 100,000 for cancers among Native Americans in Jackson and Shawnee Counties, with linear trend line.**



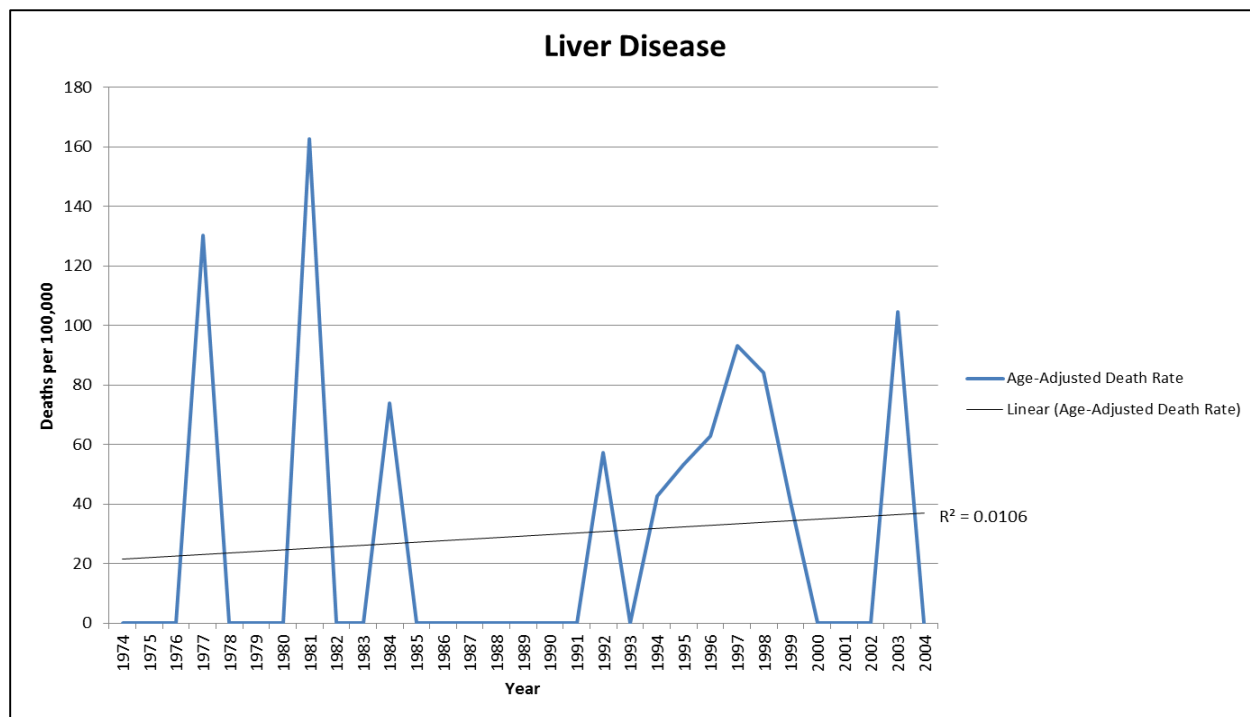
**FIGURE 8-9. Age adjusted death rates per 100,000 for strokes among Native Americans in Jackson and Shawnee Counties, with linear trend line.**



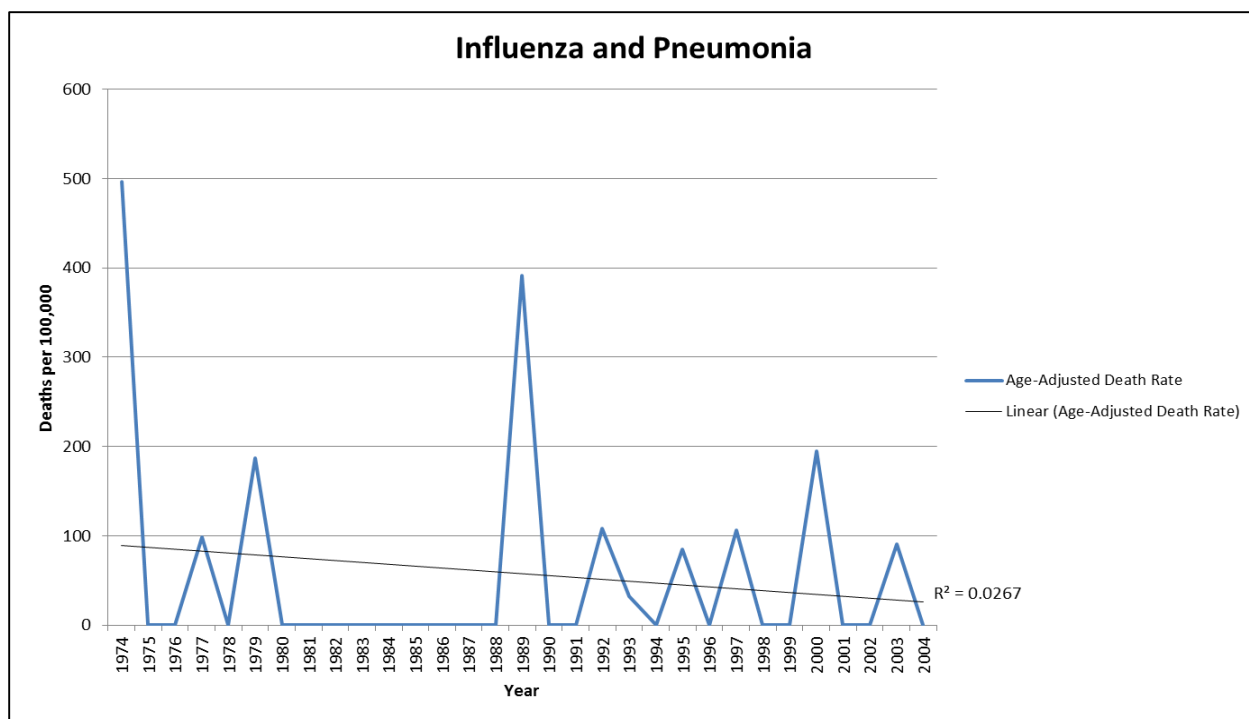
**FIGURE 8-10. Age adjusted death rates per 100,000 for accidents among Native Americans in Jackson and Shawnee Counties, with linear trend line.**



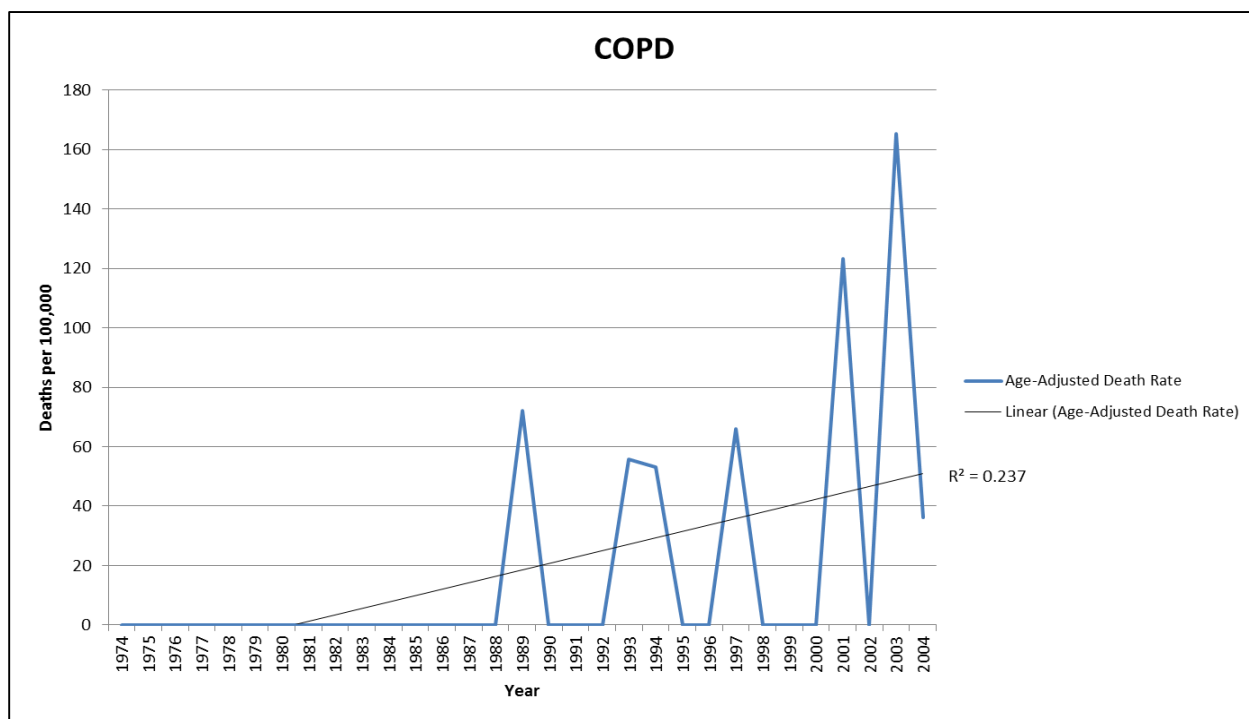
**FIGURE 8-11. Age adjusted death rates per 100,000 for diabetes among Native Americans in Jackson and Shawnee Counties, with linear trend line.**



**FIGURE 8-12. Age adjusted death rates per 100,000 for liver diseases among Native Americans in Jackson and Shawnee Counties, with linear trend line.**

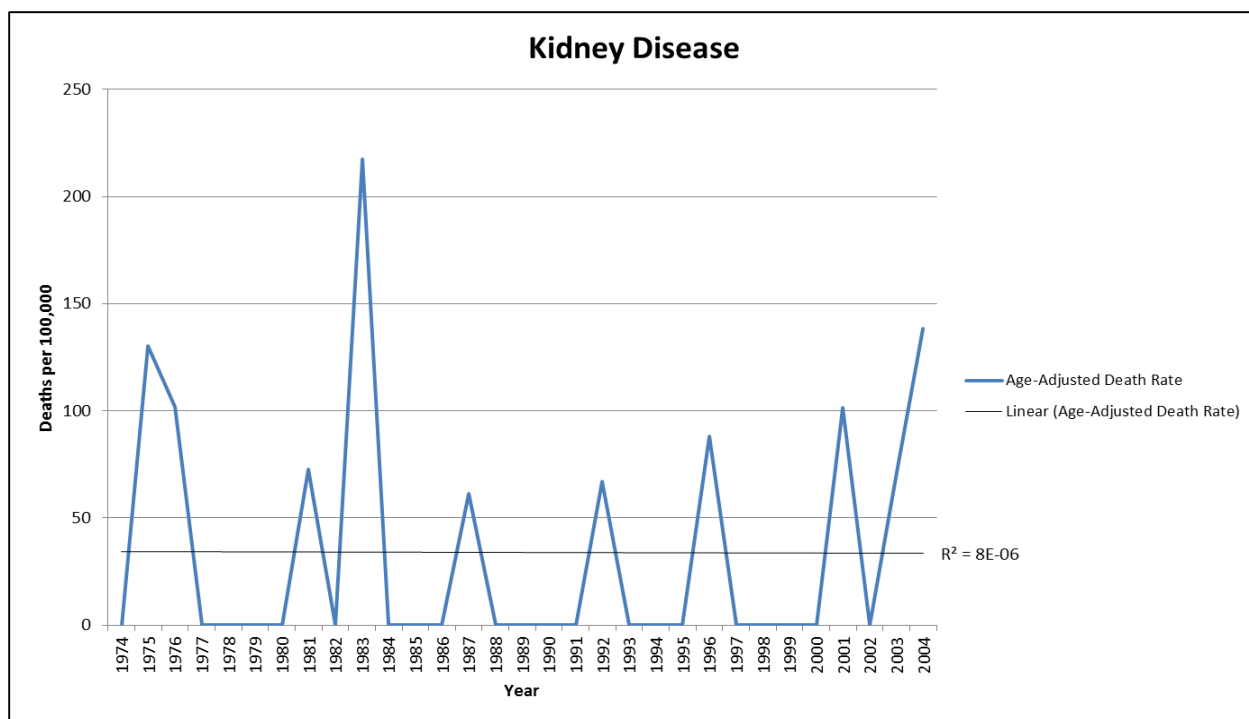


**FIGURE 8-13. Age adjusted death rates per 100,000 for influenza and pneumonia among Native Americans in Jackson and Shawnee Counties, with linear trend line.**

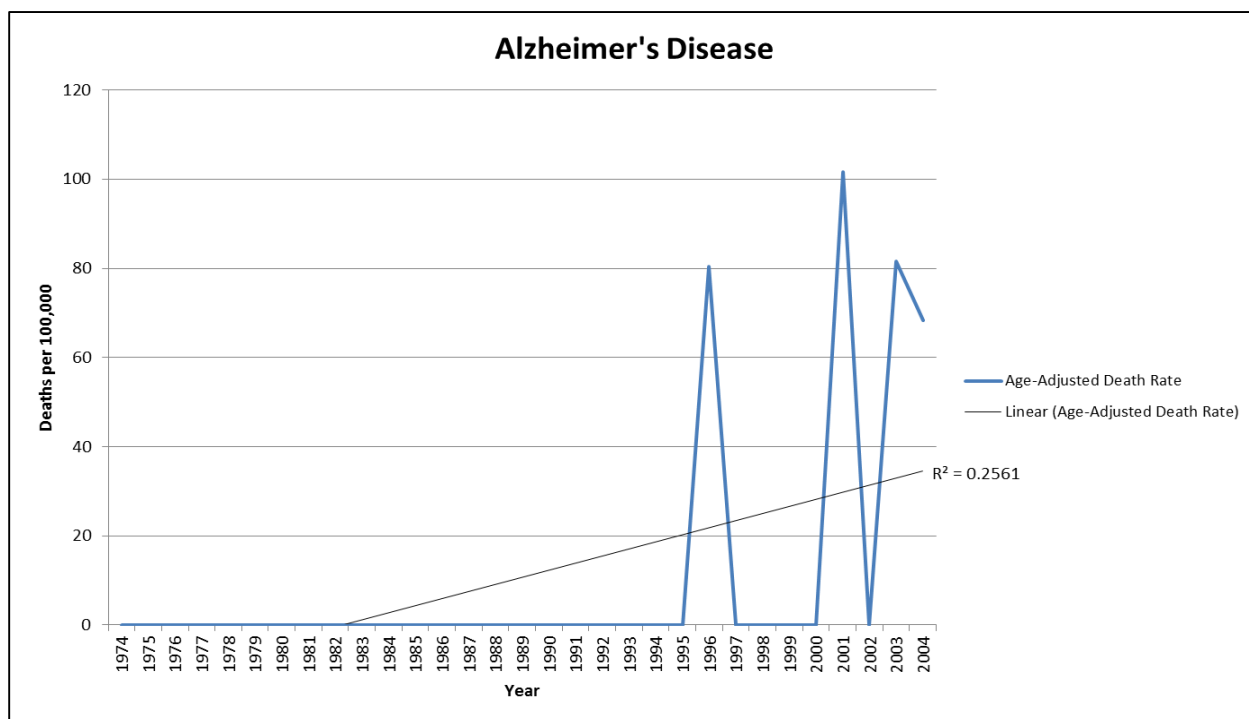


**FIGURE 8-14. Age adjusted death rates per 100,000 for COPD among Native Americans in Jackson and Shawnee Counties, with linear trend line.**

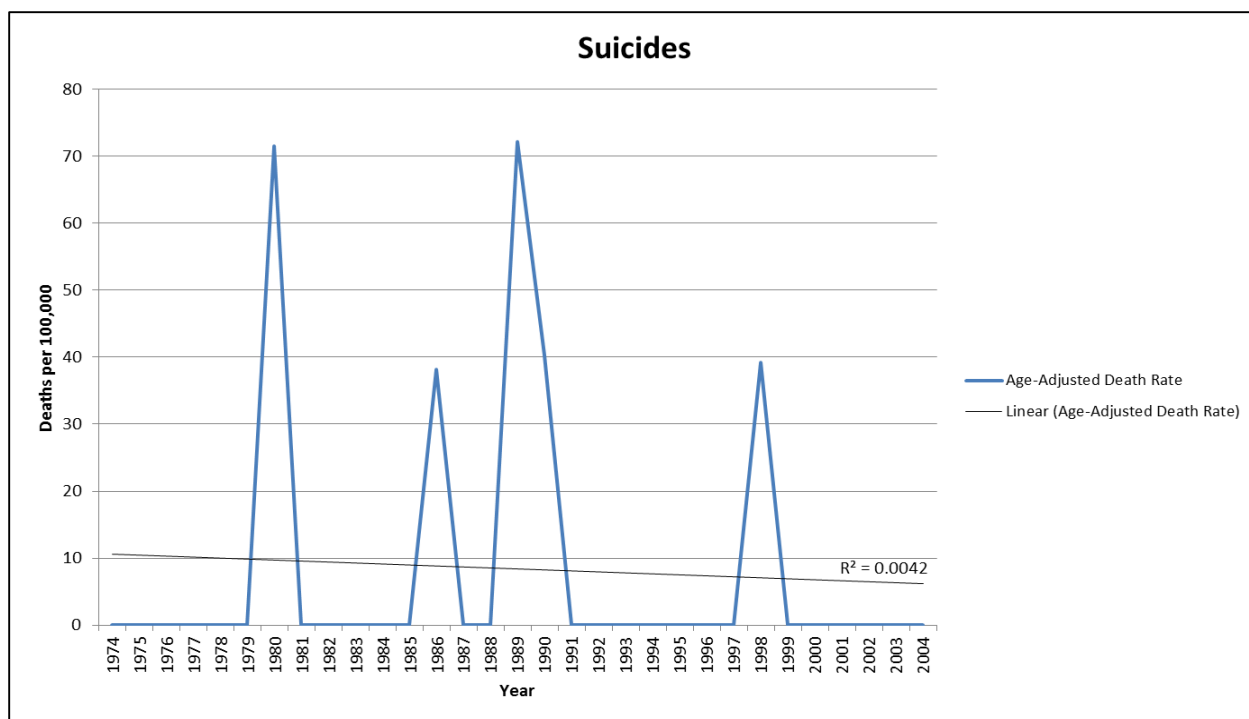




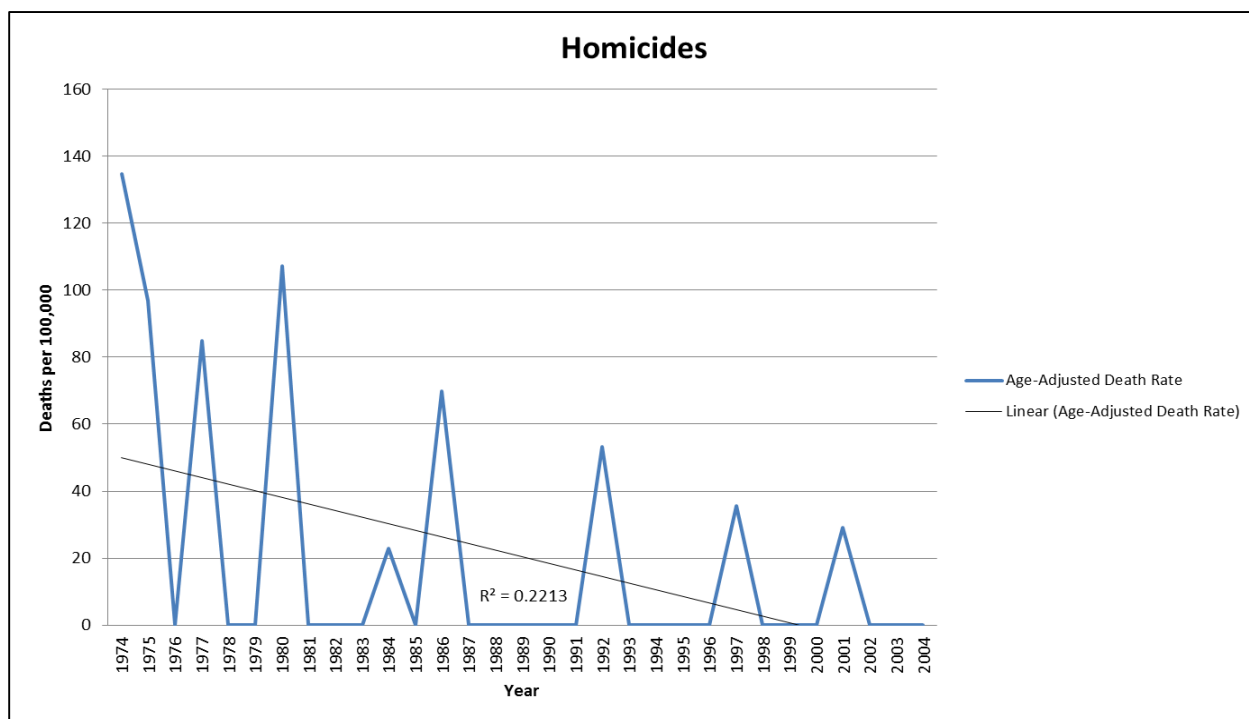
**FIGURE 8-15.** Age adjusted death rates per 100,000 for kidney disease among Native Americans in Jackson and Shawnee Counties, with linear trend line.



**FIGURE 8-16.** Age adjusted death rates per 100,000 for Alzheimer's disease among Native Americans in Jackson and Shawnee Counties, with linear trend line.



**FIGURE 8-17. Age adjusted death rates per 100,000 for suicides among Native Americans in Jackson and Shawnee Counties, with linear trend line.**



**FIGURE 8-18. Age adjusted death rates per 100,000 for homicides among Native Americans in Jackson and Shawnee Counties, with linear trend line.**

(Figure 8-8), liver disease (Figure 8-12), COPD (Figure 8-14) and Alzheimer's disease (Figure 8-16) showed upward trends.

A comparison of JK/SH crude and age-adjusted death rates *versus* the age-adjusted rates for the U.S. and all Native Americans is presented in Table 8-5. The crude death rates for the Prairie Band are comparable to the U.S. and Native American rates. After age-adjustment however, the Prairie Band rates are high for a number of chronic causes, demonstrating that the Prairie Band population is essentially younger than the standard population. Deaths due to heart disease and cancer are high, while surprisingly, deaths due to diabetes are not significantly greater than those of the general U.S. population.

**TABLE 8-5. Comparison of crude and age-adjusted mortality rates for Native Americans in Jackson and Shawnee counties from 1996 to 2004; age adjusted mortality rates for Native Americans (AI/AN), 2004 to 2006 and age-adjusted rates for the U.S in 2000. Tests of significance compare the JK/SH 1996-2004 age-adjusted death rates with those of the U.S. in 2000 (standard population) (CDC 2002; IHS 2011).**

Cause	U.S. 2000 (Age-Adjusted Death Rates)	AI/AN 2004-2006 (Age-Adjusted Death Rates)	JK/SH 1996-2004 (Crude Death Rates)	JK/SH 1996-2004 (Age-Adjusted Death Rates)	Significance
All Causes	873.1	980.0	747.6	1369.9	p<0.05
Heart Disease	258.2	206.2	206.7	388.9	p<0.05
Cancers	200.9	176.2	163.6	303.0	p<0.05
Stroke	60.9	46.6	38.8	74.6	ns
Kidney Failure	44.3		19.2	44.4	ns
Accidents	35.6	93.8	66.3	71.1	p<0.05
Diabetes	25.2	68.1	15.3	28.4	ns
Influenza and Pneumonia	23.7	27.1	19.7	43.6	p<0.05
Alzheimer's disease	18.0		15.5	36.9	p<0.05
Suicide	10.7	19.8	4.0	4.4	p<0.05
Liver Diseases	9.6		35.4	42.8	p<0.05
Homicide	6.1	11.7	7.9	7.2	ns

**TABLE 8-6. Crude mortality rankings from 2002 for racial groups (Anderson 2002), compared to rankings for Native Americans in Jackson and Shawnee counties for data from 1990-2004.**

	White	Black	American Indian	Asian/ Pacific Islander	JK/SH	White	Black	American Indian	Asian/ Pacific Islander
	Rank					Difference from JK/SH			
Diseases of heart	1	1	1	2	1	0	0	0	1
Malignant neoplasms	2	2	2	1	2	0	0	0	1
Cerebrovascular diseases	3	3	5	3	4	1	1	1	1
Chronic lower respiratory diseases	4	8	7	5	7	3	1	0	2
Accidents (unintentional injuries)	5	4	3	4	3	2	1	0	1
Influenza and pneumonia	6	10	9	6	7	1	3	2	1
Diabetes mellitus	7	5	4	7	5	2	0	1	2
Alzheimer's disease	8	13	13	13	10	2	3	3	3
Nephritis, nephrotic syndrome and nephrosis	9	9	10	9	9	0	0	1	0
Intentional self-harm (suicide)	10	14	8	8	11	1	3	3	3
Chronic liver disease and cirrhosis	11	12	6	12	6	5	6	0	6
Certain conditions originating in the perinatal period	12	11	12	10	12	0	1	0	2
Assault (homicide)	13	6	11	11	12	1	6	1	1
<b>DIFFERENCE</b>						<b>18</b>	<b>25</b>	<b>12</b>	<b>24</b>

The ranked causes of death among Native Americans in Jackson and Shawnee counties between 1990 and 2004 show that the JK/SH group is most similar to American Indians in general (Table 8-6). The largest differences in rank between the JK/SH and the greater Native American populations are for suicides, Alzheimer's disease, and influenza and pneumonia. Accidents constituted the third most common cause of death among the population between 1974 and 2004, increasing sharply between those years. The mean rate after 1990 was 8.7%. Suicides and homicides were the 11<sup>th</sup> and 12<sup>th</sup> (tie) most common causes of death after 1990, respectively. Homicides in particular showed a precipitous decline between 1974 and 2004. Suicides were less common after 1990 than during the preceding 16 years. Cancers were the second most common cause of death among Native Americans in Jackson and Shawnee counties after 1974. Cancer of the lungs was the most common among the JK/SH group since 1990, followed by

**TABLE 8-7. Cancer deaths by type for Native Americans in Jackson and Shawnee counties, 1990-2004, compared to U.S. rates, 2005 (American Cancer Society 2005).**

Cancer Type/Site	U.S. 2005	% of Cancers	JK/SH% of Cancers, 1990-2004
Brain	130	2.4	
Breast	380	7.1	1.8
Colon	610	11.4	12.3
Leukemia	230	4.3	1.8
Liver	120	2.2	1.8
Lung	1540	28.7	42.1
Non-Hodgkin Lymphoma	220	4.1	1.8
Ovary	160	3.0	5.3
Pancreas	290	5.4	1.8
Prostate	270	5.0	1.8
Stomach			8.8
Kidney			3.5
Anus			1.8
Esophagus			1.8
Major Salivary Glands			1.8
Multiple Myeloma			1.8
Nasopharynx			1.8
Rectum			1.8
Skin			1.8
Tongue			1.8
Urethra			1.8
Other	1420	26.4	1.8
TOTAL	5370	100	100

cancers of the colon, stomach, ovaries, and kidneys (Table 8-7). Lung cancer deaths made-up a much larger percentage of cancer deaths among the JK/SH community than among the general U.S. population (42.1% vs. 28.7%). The crude death rates for the JK/SH group differ by percent from those of the greater Native American population (Table 8-8). Native Americans in Jackson and Shawnee counties experience higher rates of death due to heart disease and cancers, and

**TABLE 8-8. Comparison of crude death rates by cause between Native Americans in Jackson and Shawnee counties, 1990-2004; and the total American Indian population, 2000 (Anderson 2002).**

Cause	American Indian	JK/SH
	%	%
Diseases of heart	21.0	30.3
Malignant neoplasms	17.0	19.7
Accidents (unintentional injuries)	12.0	8.7
Diabetes mellitus	5.4	4.4
Cerebrovascular diseases	5.0	5.2
Chronic liver disease and cirrhosis	4.7	4.3
Chronic lower respiratory diseases	3.8	2.6
Intentional self-harm (suicide)	2.6	1.0
Influenza and pneumonia	2.5	2.6
Nephritis, nephrotic syndrome and nephrosis	1.9	1.8
Assault (homicide)	1.8	0.8
Certain conditions originating in the perinatal period	1.1	0.8
Alzheimer's disease	0.8	1.2
Human immunodeficiency virus (HIV) disease	0.5	0.0

lower rates for accidents, diabetes, suicide, and homicide. The higher rates of heart disease among the JK/SH group appear more similar to the rates of white Americans than other Native Americans. Although they appear to differ from other Native Americans, the differences are not significant, statistically.

## Discussion

Heart disease was the most common cause of death in 1974, signifying that the epidemiologic transition had already occurred. Influenza and pneumonia, the only infectious conditions among the top causes of death, were highest in 1974. Yet, even though the rates are indicative of a population having gone through the epidemiologic transition, the Native American rates indicate substantial differences from the greater U.S. population. The age-adjusted death rate for influenza and pneumonia among the JK/SH group was nearly double that

of the U.S., showing that infectious diseases continued to take a higher toll among these Native Americans than non-natives.

Heart disease remained the primary cause of mortality among the JK/SH group between 1974 and 2004. Deaths rates due to heart disease essentially remained flat over this period of time (Fig. 8-6). Potawatomi elders recall heart attacks as a common cause of death even when they were young. One Potawatomi elder reported that in the past it seemed that heart attacks only killed the very old, but today people suffer from heart disease at much younger ages.

Strokes were less common among the JK/SH community than in the U.S., but that reflected the age structure of the JK/SH group. The age-adjusted death rate from strokes was higher among the JK/SH group, indicating an excess burden from this disease and the probability that, like other chronic conditions, the incidence will increase as the population ages.

The increasing trend in cancer mortality was the most dramatic of all the principal causes of mortality. At least 85% of lung cancers are related to cigarette smoking (Hong and Tsao 2008). The cigarette smoking rate among Native Americans is close to 40% (Daley et al. 2010). Community-based surveys indicate that the smoking rate among the JK/SH group may be comparable, at around 37%. Research in recent years also has confirmed a relationship between obesity and metabolic syndrome with cancers (Wicki and Hagmann 2011). The complex metabolic interactions involving adipose tissue, insulin, insulin-like growth factors, estrogen, leptin, adiponectin, and the metabolic pathways involving these and other factors have been implicated. The mTOR (mammalian target of rapamycin) metabolic pathway, for instance, responds to increased levels of glucose and amino acids as energy carriers, as well as to growth factors such as insulin and insulin-like growth factor. The mTOR pathway, favoring cell proliferation, has also been identified as promoting carcinogenesis (Braun, Bitton-Worms, and

LeRoith 2011; Wicki and Hagmann 2011). The symptoms of the metabolic syndrome have been linked to specific cancers. In particular, obesity has been associated with cancers of the esophagus and cardia, bile ducts, cervix, and certain hematologic cancers. Obesity, in combination with insulin resistance, has been connected to cancers of the colon, pancreas, breasts, endometrium, and kidneys. Dyslipidemia has been linked to breast, prostate, and lung cancers and non-Hodgkin lymphoma (Braun, Bitton-Worms, and LeRoith 2011). The higher rates of lung, colon, and kidney cancers observed among Jackson and Shawnee county Native Americans may reflect connections between cancer and metabolic syndrome.

In addition to obesity and metabolic syndrome, alcohol and its metabolite acetaldehyde have been determined to be carcinogenic. Acetaldehyde in particular is a Group 1 genotoxic carcinogen and is mutagenic and teratogenic as well. Studies indicate an association between alcohol intake and mouth, larynx, pharynx, esophagus, stomach, liver, and breast cancers (Wicki and Hagmann 2011; WHO 1988). Those with genetic deficiencies in alcohol dehydrogenase production are at greater risk of developing gastrointestinal and liver cancers due to greater periods of exposure to acetaldehyde from alcohol (Homann et al. 2006). Such genetic defects have been documented for Native American populations and are also implicated in higher rates of alcoholism (Mulligan et al. 2003; Reed 1985).

Diabetes was the fifth most common cause of mortality among Native Americans in Jackson and Shawnee counties after 1974, with a slight downward trend since then. The downward trend may simply be the result of a small sample size or may reflect efforts to prevent diabetes mortality through new medicines and IHS programs such as the Special Diabetes Program for Indians and the Diabetes Prevention Program. Though deaths may have been decreasing among Native Americans in Jackson and Shawnee counties. Overall Native



American rates of obesity, hypertension, and diabetes continued to increase into the 1990s (Levy, Jacober, and Sowers 1994). The Potawatomi, of the Northeast-Algonkian grouping, fall into Young's (1994) medium-prevalence group (Chapter 5, pg. 162), with a prevalence rate of 10%-30%. The incidence of diabetes has increased among Native Americans since 1995, although deaths due to diabetes decreased during this time in the JK/SH community. Improvements in diabetes treatment may account for decreasing death rates despite increasing incidence of the disease. In 1981, U.S. biotech company Genentech developed synthetic human insulin, eliminating the need to use nonhuman animals as an insulin source (Tattersall 2009). The hemoglobin A1c (HbA1c) test was developed in 1979, providing a more comprehensive view of patient blood-sugar levels over a period of two to three months (Tattersall 2009). A 10-year comprehensive study called the Diabetes Control and Complications Trial (DCCT) established the HbA1c as the standard measure for blood glucose control. The study demonstrated that those diabetics able to keep their blood-sugar levels closer to normal were less likely to develop eye, kidney, and nerve-related complications. Prior to this time most doctors did not stress tight blood-sugar control under the assumption that meticulous blood sugar monitoring and intensive insulin therapy had little impact on diabetes outcomes. The DCCT findings showed that tight blood-sugar control had positive effects for diabetics (Skyler 2012; Tattersall 2009).

In May of 1995, metformin, an oral medication for people with type 2 diabetes and derived from the French lilac or goat's rue (*Galega officinalis*), was finally approved for use in the United States by the FDA. Unlike sulfonylurea drugs, which stimulate insulin release, metformin does not increase insulin production. Instead, it heightens sensitivity to insulin and increases the muscles' ability to use the insulin. Since metformin promotes weight loss, decreases hyperglycemia, and improves lipid levels, it has been shown to be an effective tool for

people with type 2 diabetes when used in conjunction with sulfonylureas (Bailey and Day 2004; Skyler 2012). The glitazones were also introduced as another class of insulin-sensitizing drugs in the 1990s (Daniel 2000).

Congress first appropriated funds for the Special Diabetes Program for Indians, administered through the IHS, in 1997. Deaths from diabetes among the JK/SH showed the highest peaks prior to this time. The program has had a dramatic effect on preventive services for Native Americans (Table 8-9). The trend for diabetes deaths, however, was downward prior to the SDPI, so other factors are likely contributors. New drug therapies, intensive glucose control, and an emphasis on healthier behaviors all contributed to better diabetes management and prevention of deaths directly due to the condition.

Social pathologies comprised a substantial proportion of deaths. Age-adjusting the accident death rates reduced the rank of accidents to fourth, consistent with the younger age of the JK/SH population and the tendency for accidents to claim younger people. Accidents, suicides, and homicides were the leading causes of death among Native Americans under 20 years old. Motor vehicle crashes were responsible for the largest proportion of accidents among Native Americans between 1989 and 1998. Auto/pedestrian accidents, firearms accidents, and

**TABLE 8-9. Native American access to diabetes treatment and prevention services after the implementation of the SDPI (IHS 2012).**

Service	1997	2010
Diabetes clinics	31%	71%
Diabetes clinical teams	30%	94%
Diabetes patient registries	34%	94%
Nutrition services for adults	39%	89%
Access to registered dietitians	37%	77%
Culturally tailored diabetes education programs	36%	99%
Access to physical activity specialists	8%	74%
Adult weight management programs	19%	76%

drownings were the next most common accident categories (IHS 1998). The community experienced lower rates of homicides and suicides than the total Native American population; suicide and murder rates since 1990 were less than half that of the larger Native American population.

As mentioned above, cigarette smoking was a likely contributor to the high lung-cancer mortality. Alcohol use was also a significant contributor to deaths. Among liver diseases – the seventh most common cause of death among the JK/SH community – those resulting from alcoholic liver disease constituted 73% of the total. Liver diseases were over four times greater among Native Americans in the Jackson and Shawnee counties from 1990 – 2004 than among the general U.S. population in 2000. Compared to other Native Americans, the JK/SH group had a slightly lower rate. Alcohol is also known to be a primary contributor to automobile accidents, and automobile accidents were the primary cause of alcohol-attributable deaths among Native Americans between 2001 and 2005 (CDC 2008).

As described in Chapter 5, alcohol consumption (Singh and Hoyert 2000), cigarette smoking (Flint and Novotny 1997) and other social pathologies (Trovato 1988) are related to poverty and unemployment. However, among the JK/SH community, deaths due to social pathologies were lower than those of Native American populations across the nation, although the poor economic situation of the Potawatomi during the first part of the 20<sup>th</sup> century continued into the middle of the century. Forty-seven Potawatomi families received county or state financial assistance in 1951 (Kelly 1952). Many older Potawatomi reported leaving the reservation as young adults, and their reasons for leaving included the lack of government assistance and the lack of jobs on the reservation. A new governmental constitution in 1961 was supposed to provide them with greater control over their own affairs and improve their cultural

and economic living conditions. However, internal power struggles continued to interfere with the tribe's ability to focus on economic development (Clifton 1998). In 1965 homes were small two- or three-room single story wood-frame houses—most with electricity but without indoor plumbing (Searcy 1965). Most of these homes had been lived in for decades. Telephones were scarce, although many families had a car. In 1967 Prairie Band members began to receive per capita payments from the interest of a claims settlement over the 1846 Prairie du Chien Treaty totaling \$1,176,788. Each tribal member received \$490.50, a windfall with little lasting impact on the recipients' socioeconomic status. By 1975 the Prairie Band had yet to reap any economic benefits from the new constitution and had accomplished little socioeconomically (Clifton 1998).

In the early 1970s, Lester Jessepe (1973) reported that unemployment at the Prairie Band Reservation was as high as 50%, and that the high school dropout rate was 44%. The Indian Self-Determination Act promised economic relief to tribes through various federal grants, but civil unrest on the Prairie Band Reservation against the U.S. government led the BIA to suspend the tribal constitution and deprived the Prairie Band of federal funding (Mitchell 1995). Reservation residents recall the 1970s and 1980s as periods of poverty, with many people still living in homes without indoor plumbing and some still lacking electricity. After the new tribal constitution was approved by the BIA in 1977, the tribe again was able to benefit somewhat from federal funding, but the benefits were short-lived and eventually cut by the Reagan administration, extending the economic slump on the reservation. Not until 1987, when the tribe assumed control of the bingo operation on the reservation, did the Prairie Band experience an extended period of economic growth (Mitchell 1995).

Poverty likely played a role in the epidemiologic pattern displayed by the tribe into the 1980s. While well into the third epidemiologic transition, the JK/SH community did display a

peculiar morbidity pattern, with middle-aged individuals experiencing a high number of health problems. According to an analysis of the health of the Prairie Band Potawatomi in 1983 (John 1986), the most commonly reported health problems were arthritis, vision problems, hearing problems, and diabetes. Among the Prairie Band elders aged 65 and over, diabetes, hypertension, arthritis, ischemic heart disease, and congestive heart failure were the medical problems most often prompting doctor visits. Diabetes, hypertension, and arthritis were the most common medical problems among those over age 45. At this time, Potawatomi patients in the age group 45-64 made up a larger percentage (41%) of those patients with 14 or more doctor office visits per year than did those in the 65+ age group (37%). Increased morbidity is associated with lower incomes, most evident below the poverty level up to near the median income; thereafter increasing income provides no further morbidity reduction. Good health is strongly associated with higher income and higher educational attainment (Norris et al. 2003).

In 1998 the Prairie Band opened a casino on the reservation, managed by Harrah's Entertainment, Inc. The casino has had a dramatic economic impact on the reservation as well as Jackson and Shawnee counties. The Prairie Band used much of the revenue from gaming for infrastructure and community services. Roads were improved, and new buildings were constructed for the tribal government, a Boys & Girls Club, an Early Childhood Development Center, an elder center, the police department, and fire and emergency medical services. Housing clusters, including a section specifically for elders, were also constructed. Since the casino was opened, the tribal council has mandated that 43% of revenues go toward the nation's economic development and infrastructure needs (Heck 2007a).

The income impact of the casino on Jackson and Shawnee counties was estimated to be around \$39 million in 1999. Twenty-five million dollars of this income was distributed to

households as payroll. The largest percentage of this payroll (\$21,325 million) went to employees living in Jackson and Shawnee counties. Sixty-two of these employees lived on the reservation, but 382 lived elsewhere in Jackson County, 487 resided in Shawnee County, and 21 lived in Brown County (Darling, Yang, and Ariyaratne 2001). By 2002, the number of employees had dropped slightly to 937, who received wages and benefits equaling \$17.4 million (Seitz and Darling 2003). In 2007 the tribe assumed full control of the casino from Harrah's. Currently, the number of employees of the casino is down to 800, approximately 120 of whom are tribal members (Prairie Band Potawatomi Nation 2011). A General Council vote in 2007 increased the per capita distribution payouts to tribal members from 30% to 48% (Heck 2007b). Quarterly payouts increased to approximately \$1,500 per member.

Some tribes experience poverty rates as high as 40% (Sarche and Spicer 2008). In contrast, the poverty rate of Native Americans living in Jackson County, Kansas, in 2009 was 15.1% (City-Data.com 2011), only slightly higher than the state poverty rate of 13.2% (Bureau of the Census 2011). For Native Americans in Jackson County, Kansas, Census Bureau estimates indicate a high school graduate rate of nearly 92% (2005-2009 data), which compares favorably with the overall county graduation rate of 90% (2006-2007 data). A bachelor's degree attainment rate of 10.8% is given for Native Americans in Jackson County (Bureau of the Census 2010; County Health Rankings 2011). Compared to the circumstances of some tribes, the Native Americans of the Prairie Band community show a more equitable socioeconomic position in relation to the surrounding non-Indian communities.

## **Conclusions**

For the time period 1974-2004 chronic conditions were high, and cancer was on the increase. Infectious conditions were declining, but remained higher than national averages.

Deaths related to social pathologies were also high, but lower than for many other Native American groups. The aging of the population may explain the increases in deaths due to chronic diseases such as cancer and COPD, and reduction of deaths due to social pathologies such as homicides, which tend to impact younger people to a greater degree. An increase in the mean age at death also points to an aging population, with older individuals accounting for more deaths. Increases in Alzheimer's disease and COPD may simply reflect the lack of deaths due to these conditions before the late 1980s, or to increasing attention to these conditions in the health care sector. An aging demographic profile is consistent with a population in the *Age of Degenerative and Man-Made Diseases* (Omran 1977). As the Native Americans in Jackson and Shawnee counties continue to age, the burden of chronic conditions may be expected to take an increasing toll.

The period 1974-2004 was also one of increasing affluence for the tribe, particularly toward the end of the century. This increasing affluence could be another contributor to the observed epidemiologic pattern. The epidemiologic profile of the Native Americans in Jackson and Shawnee counties indicates lingering disparities compared to the greater U.S. population, reflecting historical poverty and continued economic disadvantages. Yet the lower death rates for those causes related to social pathologies – accidents, suicides, and homicides – compared to other Native Americans suggests that the Native Americans in Jackson and Shawnee counties have been able to improve the quality of life for tribal members in recent years, reducing the levels of infectious diseases and limiting social pathologies, relative to other Native American tribes. Revenues from casino gaming have undoubtedly provided the tribe with resources to provide greater services and jobs to tribal members. Increased income also is likely to have directly contributed to the high rate of chronic conditions. An increase in income provided a

greater ability to purchase high-fat, high-calorie fast food and restaurant foods, contributing to obesity and the diseases associated with the condition. However, poverty is a legacy of reservation life, and the persistence of higher rates of poverty in the community contributes to the persistence of health disparities.

## Chapter 8 References Cited

- American Cancer Society. 2005. *Cancer Facts and Figures 2005*. Atlanta: American Cancer Society.
- Anderson, R.N. 2002. "Deaths: Leading Causes for 2000," in *National Vital Statistics Reports*, 50(16). Hyattsville, Maryland: National Center for Health Statistics.
- Arias, E., Schauman, W.S., Eschbach, K., Sorlie, P.D., Backlund, E. 2008. "The Validity of Race and Hispanic Origin Reporting on Death Certificates in the United States," in *National Center for Health Statistics. Vital and Health Statistics*, 2(148). Washington, DC: U.S. Government Printing Office.
- Bailey, C.J. and Day, C. 2004. Metformin: Its Botanical Background. *Practical Diabetes International* 21(3):115-117.
- Braun, S., Bitton-Worms, K., and LeRoith, D. 2011. The Link Between the Metabolic Syndrome and Cancer. *International Journal of Biological Sciences* 7(7):1003-1015.
- Bureau of the Census. 2010. 2005-2009 American Community Survey 5-Year Estimates. [http://factfinder.census.gov/servlet/DataSetTableListServlet?\\_ds\\_name=ACS\\_2009\\_5YR\\_G00\\_&\\_type=table&\\_program=ACS&\\_lang=en&\\_ts=338922384416](http://factfinder.census.gov/servlet/DataSetTableListServlet?_ds_name=ACS_2009_5YR_G00_&_type=table&_program=ACS&_lang=en&_ts=338922384416). June 6<sup>th</sup>, 2010.
- . 2011. State & County QuickFacts. <http://quickfacts.census.gov/qfd/states/20/20085.html>. October 28<sup>th</sup>, 2011.
- CDC (Centers for Disease Control and Prevention). 2002. LCWK9. Deaths, percent of total deaths, and death rates for the 15 leading causes of death: United States and each State, 2000. National Vital Statistics System, National Center for Health Statistics. [http://www.cdc.gov/nchs/data/dvs/LCWK9\\_2000.pdf](http://www.cdc.gov/nchs/data/dvs/LCWK9_2000.pdf). December 13<sup>th</sup>, 2011.
- . 2008. Alcohol-Attributable Deaths and Years of Potential Life Lost Among American Indians and Alaska Natives --- United States, 2001—2005. *Morbidity and Mortality Weekly Report* 57(34):938-941.
- City-Data.com. 2011. Jackson, County, Kansas (KS). [http://www.city-data.com/county/Jackson\\_County-KS.html](http://www.city-data.com/county/Jackson_County-KS.html). October 28<sup>th</sup>, 2011.
- Clifton, J.A. 1998. *The Prairie People: Continuity and Change in Potawatomi Indian Culture, 1665 - 1965*. Iowa City: University of Iowa Press.



County Health Rankings. 2011. 2011 Jackson, Kansas - High school graduation. <http://m.countyhealthrankings.org/node/1065/21>.

Curtin, L.R. and Klein, R.J. 1995. "Direct Standardization (Age-Adjusted Death Rates)," in *Healthy People 2000 Statistical Notes*. Centers for Disease Control and Prevention, National Center for Health Statistics. Hyattsville, Maryland: U.S. Department of Health and Human Services.

Daley, C.M., Greiner, A.K., Nazir, N., Daley, Solomon, C.L., Braiuca, S.L., Smith, T.E., and Choi, W.S. 2010. All Nations Breath of Life: Using Community-Based Participatory Research to Address Health Disparities in Cigarette Smoking among American Indians. *Ethnicity & Disease* 20:334-338.

Daniel, K. 2000. The Glitazones: Proceed with Caution. *Western Journal of Medicine* 173(1):54-57.

Darling, D.L., Yang, X., and Ariyaratne, C.B. 2001. The Role of the Prairie Band Casino Business in the Area Economy: Jackson and Shawnee Counties in 1999. *Kansas Business & Economic Review* 24(1):19-24.

Flint, A.J. and Novotny, T.E. 1997. Poverty Status and Cigarette Smoking Prevalence and Cessation in the United States, 1983-1993: The Independent Risk of Being Poor. *Tobacco Control* 6:14-18.

Heck, S. 2007a. Nation Taking Charge of its Own Destiny: July 1 Casino Management Takeover Announced at Press Conference. *Prairie Band Potawatomi News*, April 2007 Edition, 1.

—. 2007b. PBP Directors of Departments and Programs Bracing for 2008 Budget Cuts. *Prairie Band Potawatomi News*, November 2007 Edition, 1.

Heron, M., Hoyert, D.L., Murphy, S.L., Xu, J., Kochanek, K.D., and Tejada-Vera, B. 2009. Deaths: Final Data for 2006. *National Vital Statistics Reports* 57(14). Hyattsville, MD: National Center for Health Statistics.

Homann, N., Stickel, F., König, I.R., Jacobs, A., Junghanns, K., Benesova, M., Schuppan, D., Himsel, S., Zuber-Jerger, I., Hellerbrand, C., Ludwig, D., Caselmann, W.H., and Seitz, H.K. 2006. Alcohol Dehydrogenase 1C\*1 Allele is a Genetic Marker for Alcohol-Associated Cancer in Heavy Drinkers. *International Journal of Cancer* 118:1998-2002.

Hong, W.K. and Tsao, A.S. 2008. "Lung Carcinoma" in *The Merck Manual for Health Care Professionals*. [http://www.merckmanuals.com/professional/pulmonary\\_disorders/tumors\\_of\\_the\\_lungs/lung\\_carcinoma.html?qt=&sc=&alt=](http://www.merckmanuals.com/professional/pulmonary_disorders/tumors_of_the_lungs/lung_carcinoma.html?qt=&sc=&alt=). March 1<sup>st</sup>, 2008.

IHS (Indian Health Service). 1998. *Trends in Indian Health, 1997*. Rockville, MD: U.S. Department of Health and Human Services.

—. 2011. Indian Health Disparities. IHS Fact Sheets, <http://www.ihs.gov/PublicAffairs/IHSBrochure/Disparities.asp>. December 13<sup>th</sup>, 2011.

—. 2012. Special Diabetes Program for Indians: Community-Directed Successful Interventions and Sustained Achievements. [http://www.ihs.gov/MedicalPrograms/Diabetes/HomeDocs/Resources/FactSheets/2012/Fact\\_Sheet\\_SuccessInterv\\_508c.pdf](http://www.ihs.gov/MedicalPrograms/Diabetes/HomeDocs/Resources/FactSheets/2012/Fact_Sheet_SuccessInterv_508c.pdf). April 18<sup>th</sup>, 2012.

- Jessepe, L.L. 1973. "How to Survive When the Government Tries to Steal Everything You Have." in *Our Story: The Prairie Band Potawatomi Indians*. Topeka, Kansas.
- John, R. 1986. *Aging in a Native American Community: Service Needs and Support Networks among Prairie Band Potawatomi Elders*. PhD Dissertation, The University of Kansas.
- Kelly, C.G. 1952. *Welfare Aspects of the Potawatomi Indian Agency*. Master's Thesis, The University of Kansas.
- Levy, M.T., Jacober, S.J., and Sowers, J.R. 1994. Hypertensive Disorders of Pregnancy in Southwestern Navajo Indians. *Archives of Internal Medicine* 154(19):2181-2183.
- Mitchell, G.E. 1995. Stories of the Potawatomi People: From Early Days to Modern Times. Originally published in the Topeka Capital Journal. <http://www.kansasheritage.org/pbp/books/mitch/mitchbuk.html>
- Mulligan, C.J., Robin, R.W., Osier, M.V., Sambuughin, N., Goldfarb, L.G., Kittles, R.A., Hesselbrock, D., Godlman, D., and Long, J.C. 2003. Allelic Variation at Alcohol Metabolism Genes (ADH1B, ADH1C, ALDH2) and Alcohol Dependence in an American Indian Population. *Human Genetics* 113:325-336.
- Norris, J.C., van der Laan, M., Lane, S., Anderson, J.N., and Block, G. Nonlinearity in Demographic and Behavioral Determinants of Morbidity. *Health Services Research* 38(6 part 2):1791-1818.
- Omran, A. 1977. A Century of Epidemiologic Transition in the United States. *Preventive Medicine* 6(1):30-51.
- Prairie Band Potawatomi Nation. 2011. Prairie Band Casino. Edited by S. Heck. October 24, 2011 <http://www.pbpindiantribe.com/prairie-band-casino.aspx>
- Reed, T.E. 1985. Ethnic Differences in Alcohol Use, Abuse, and Sensitivity: A Review with Genetic Interpretation. *Social Biology* 32:195-209.
- Sarche, M. and Spicer, P. 2008. Poverty and Health Disparities for American Indian and Alaska Native Children: Current Knowledge and Future Prospects. *Annals of the New York Academy of Sciences* 1136:126-136.
- Searcy, Ann M. 1965. *Contemporary and Traditional Prairie Potawatomi Child Life*. KU Potawatomi Study Research Report Number 7. Lawrence, Kansas: Department of Anthropology.
- Seitz, M. and Darling, D.L. 2003. *The Role of Harrah's Prairie Band Casino Property in the Area Economy: Jackson and Shawnee Counties in 1998 – 2001, C.D. Study Report #212, Final Report*. K-State Research and Extension, Department of Agricultural Economics, Kansas State University.
- Singh, G.K., and Hoyert, D.L. 2000. Social Epidemiology of Chronic Liver Disease and Cirrhosis Mortality in the United States, 1935 – 1997: Trends and Differentials by Ethnicity, Socioeconomic Status, and Alcohol Consumption. *Human Biology* 72(5):801-820.
- Skyler, J.S. 2012. *Atlas of Diabetes*. New York: Springer.
- Tattersall, R. 2009. *Diabetes: The Biography*. New York: Oxford University Press.

Trovato, F. 1988. Mortality Differentials in Canada, 1951-1971: French, British, and Indians. *Culture, Medicine and Psychiatry* 12:459-477.

WHO (World Health Organization). 1988. Alcohol Drinking: Summary of Data Reported and Evaluation. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 44*. Lyon, France: International Agency for Research on Cancer.

Wicki, A. and Hagmann, J. 2011. Diet and Cancer. *Swiss Medical Weekly* 141:1-8.

Young, T.K. 1994. *The Health of Native Americans: Toward a Biocultural Epidemiology*. Oxford: Oxford University Press.

## Chapter 9: Conclusions

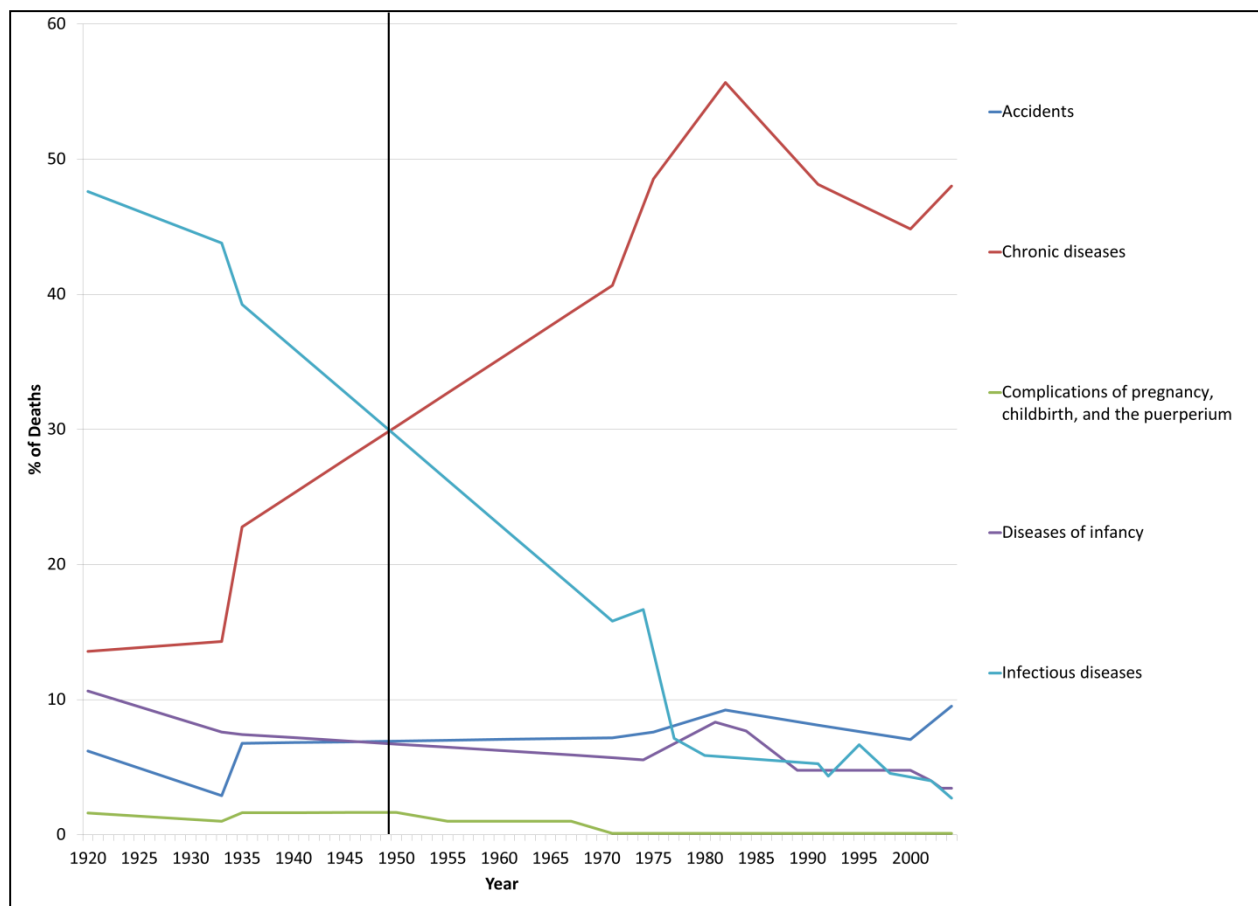
This final chapter links the information from the previous chapters to create a comprehensive view of the epidemiologic transition among the Prairie Band Potawatomi through the 20<sup>th</sup> century. Data from the early part of the century (Chapters 6 and 7) are contrasted with more recent data (Chapter 8) to elucidate temporal mortality changes. These changes, when combined with data from Chapter 5, indicate approximately when the epidemiologic transition occurred. Recent mortality trends among the Prairie Band and other Native American groups show that their epidemiologic transition differs from that of the larger U.S. population. Explanations for the observed differences draw from the issues discussed in Chapter 4.

Few data exist for morbidity or mortality among the Prairie Band Potawatomi during the middle part of the 20<sup>th</sup> century. The nature of Native American health data is such that a large amount of data is available for the end of the century and a smaller amount of data at the beginning, with a dearth of records for the decades in between (Carter and Sutch 2006). Such data gaps make it difficult to characterize mortality trends, particularly among small tribes garnering little federal attention. Additionally, the data that do exist for Native Americans in states such as Kansas consist of small samples from small populations, making statistical tests and identification of trends problematic. Table 9-1 shows the small number of deaths recorded for Native Americans in Kansas from 1960 to 1971. Even today, the Prairie Band Potawatomi do not track deaths as a part of their clinic database. Only now are they working with the Kansas Department of Health and Environment to get this information, in the manner similar to that used in this study.

**TABLE 9-1. Total number of Indian deaths in Kansas, 1960-1971 (IHS 1974).**

	1960	1961	1962	1963	1964	1965	1966	1967	1968	1969	1970	1971
Kansas	21	27	25	27	23	22	23	26	43	39	-	48

Data available for the early part of the 20<sup>th</sup> century indicate that infectious diseases were the most common causes of morbidity and mortality among the Prairie Band, with trachoma and tuberculosis most prominent. By the last quarter of the century, chronic conditions had become by far the most common causes of death. In addition, accidents and deaths related to social pathologies become more common. Based on the data presented in Chapters 6, 7, and 8, mortality from chronic diseases began to exceed that from infectious diseases in 1949 (Figure 9-1), the same year this trend began among other Native Americans (see Figure 5-3, pg. 157). This marks the time at which the Prairie Band entered the *Age of Degenerative and Man-Made Diseases*, much later than for the general U.S. population. The transition was delayed among



**FIGURE 9-1. Mortality transition among the Prairie Band community.**

Native Americans such as the Prairie Band due to the conditions on reservations caused by the poverty that resulted from allotment and governmental policies. Federal attention to these deplorable conditions did not occur until the Merriam Report in 1928, and serious efforts to alleviate the suffering were not enacted until the 1930s.

The poor conditions facing Native Americans at the beginning of the 20<sup>th</sup> century were a part of the reservation policy established by the 1851 Indian Appropriations Act. Resettlement on reservations limited the ability of tribes to effectively practice their traditional subsistence and procure adequate food. The traditional diet of the Potawatomi had consisted of lean meat from wild game and fish, wild plants, and cultivars, including corn, beans, and squash. Much of this food was dried or smoked for use throughout the winter (Edmunds 1978).

In the early part of the 20<sup>th</sup> century, the Potawatomi were compelled to abandon their traditional lifeways and adopt the culture of the larger U.S. society. Indian commissioners saw a positive relationship between farming and better health (Putney 1980). Commissioner Sells wrote that farming led to better nutrition for the Indians and should be a principal tool in preventive medicine (Sells 1919). Indeed, there is evidence to suggest that more food and improved nutrition played a significant role in the eventual reduction of tuberculosis on Indian reservations (Putney 1980), albeit the reservation system was a major cause of malnutrition and endemic TB in the first place. Ironically the imposition of this subsistence strategy on Native Americans made them dependent on foods now implicated in high rates of obesity and diabetes (Neel, Weder, and Julius 1998). Also, farming kept them poor, relative to other segments of U.S. society (McElvaine 1984). Despite the social destruction caused by confinement on reservations and disruption of traditional subsistence, severe starvation or wasting was not recorded among the Potawatomi in the 20th century. This was likely the result of interventions

to prevent disease outbreaks by providing food commodities. Additionally, the Prairie Band produced their own food throughout much of the century via gardening and hunting.

Reservations also encouraged the spread of infectious diseases by creating crowded, unsanitary living conditions. Infectious diseases continued to be a major problem for Native Americans into the middle of the century in spite of eradication efforts. The Indian Service still considered tuberculosis to be a major health problem into the early 1950s. At the Potawatomi Agency however, only twelve out of 436 individuals tested positive for TB in 1949, with only one active case. There had been no deaths due to TB on the reservation for the previous five years (Kelly 1952), although living Potawatomi elders do recall TB as being a health scourge when they were young.

## **Modernization**

The reservation system and, later, allotment, served to create the poor health status of Native Americans during the first part of the 20<sup>th</sup> century. Assimilation, termination, and self-determination policies were supposed to alleviate the problems caused by the earlier policies, but instead contributed to the significant disparities in chronic diseases in the modern age.

There have been a number of dramatic changes in Indian health and demographics since the inception of the Indian Health Service in 1955. The containment of infectious and communicable diseases such as tuberculosis and trachoma has made a significant positive impact on health (IHS 2005). Public health measures, immunizations, increased food availability, and eventual improvements in living standards contributed to this decline in infectious diseases. Despite high rates of infant mortality throughout much of their history (Berry et al. 2004; IHS 2004), Native Americans have experienced steep declines in infant deaths in recent decades (Rhoades 2003; Young 1996). A significant reduction in deaths during infancy and childhood

has been the primary contributor to the improvement in life expectancy among Native Americans (Young 1994). The most significant decrease in mortality has been due to the reduction in tuberculosis and gastroenteritis (Young 1996).

Omran recognized that as populations moved into the *Age of Degenerative and Man-Made Diseases*, the most dramatic changes in disease patterns occurred among young women and children. Infectious diseases took a heavy toll on immunologically vulnerable infants, as well as on puerperal mothers so easily exposed to pathogens (Omran 1971). Infant deaths were among the most significant contributors to Native American mortality in the early part of the 20<sup>th</sup> century (Townsend 1938). Diseases of infancy were responsible for nearly 8% of deaths among the Prairie Band between 1924 and 1933. Disparity in infant death rates between Native Americans and the rest of the U.S. decreased markedly after 1955. Childbirth was the fourth most commonly cited cause of death among the Prairie Band in the 1928 survey. Maternal mortality rates among Native Americans also began dropping significantly in the 1950s (Hill and Spector 1971). By the late 1960s midwife services were no longer utilized on the Prairie Band reservation. By this time most Indian babies were born in modern hospitals, usually the hospital in Holton (Howell 1970). Mortality reduction among mothers and infants was due to the application of modern hygiene, use of antiseptics, and the advent of antibiotics, available in hospitals.

The changing socioeconomic conditions of modernization factor into progress in health conditions among Native Americans. Only about 16% of Indians were living in urban areas in 1950. Poverty on rural reservations and the relocation policy during the termination period contributed to 60% of Indian people living in urban areas by the year 2000. Approximately 67% of Indian people experienced overcrowded housing and lacked plumbing, electricity, and



sufficient sanitation in 1950. As a consequence of migration to urban areas and improved conditions on reservations, overcrowding was reduced to 15% by 2000. Over a quarter of a million Indian households received proper sanitation following the 1959 Indian Sanitation Facilities Act. The number of Indian households with indoor plumbing increased fourfold between 1950 and 2004. The median number of years in school for Indians was 7.1 in 1950; by the year 2000 over 70% of Indians had at least a high school diploma. Median annual income among Indian people in 1950 was \$725, roughly 16% of the income they would have by 1999 (IHS 2005).

The years following World War II saw dramatic change across all of America. Farming continued to decline, and more people relocated to urban areas seeking wage labor (French 1997). As in much of the rest of the U.S., Native Americans relocated to urban areas where employment would be more widely available. In this, they were encouraged by the government's termination policy. This relocation sped the rate of assimilation, immersing many Native Americans into the larger culture just as American concepts of work, food, housing, transportation, and consumption modernized.

An aspect of modernization that significantly impacts health is change in diet. The nutrition transition as experienced by the Prairie Band and other tribes is an accelerated version of that described by Popkin (1993). The shift to urban living and greater availability of urban products favored a diet featuring more refined grains, sugar and fat, and emphasizing highly processed foods. The food industry, media, and the government played a greater role in determining their diet as Native Americans modernized and improved socioeconomically. Changes in their socioeconomic status also led to shifts in women's domestic roles and associated shifts in the family's dietary intake. These characteristics are typical of Popkin's

Western high-income model of nutrition transition (1993), but among Native Americans the transition was accelerated and occurred primarily after World War II. It also featured food insecurity as a component of the reservation period, prompting Native Americans to invent high calorie, filling foods such as fry bread from meager government rations. Boarding schools forced Indian children to abruptly adopt a completely foreign diet, as did military service. This history shows that dietary intake shifted radically both in nutritional composition and energy content over the course of just a few generations, accompanying shifts in mortality.

Searcy (1965) did not report any cases of obvious undernutrition among the children on the Potawatomi Reservation in the mid-1960s. In fact, she reported a greater tendency for the children to be plump as opposed to thin, although non-healing wounds, eye infections, dull hair and skin tones, and listlessness among the children of poorer families were possibly results of an inadequate diet. One Potawatomi elder described herself and her siblings as "...always on the chunky side" as children. In the mid-1960s the Potawatomi diet consisted primarily of starches: white rice, white bread, corn, beans, and potatoes were the staples. A typical meal consisted of boiled rice with a small amount of meat, boiled potatoes, fried bread, and coffee. Families that kept chickens had access to meat and eggs; but those that did not had little access to animal protein. Some tribal members now over the age of 30 who grew up on the reservation reported being too poor when they were young to purchase much in the way of fast foods. The situation changed for most Potawatomi after the casino was built in 1998, which provided jobs and regular per-capita income payments to tribal members and provided income for purchasing more restaurant foods.

Modernization in America since 1950 has also been characterized by decreasing physical activity associated with changes in wage labor, housework and domestic activities, and

transportation. Increasing time spent on low physical activity pursuits such as television viewing, eating, and using computers, has been documented (Brownson and Boehmer 2004). Televisions, nearly ubiquitous in American homes today (98%), were in only 10% of homes in 1950 (Putnam 1995). Native Americans experiencing relocation during the termination period went from 19<sup>th</sup> century-like conditions on reservations to those characteristic of modernizing post-war American cities. By the end of the century, the self-reported incidence of physical inactivity among Native Americans was 32.5% (CDC 2003). Among the Native Americans recruited for the Kansas Coalition's (Potawatomi and Kickapoo reservations, and Haskell IHS in Lawrence) Diabetes Prevention Program in 2006, 48% reported engaging in no regular physical activity.

### **Current Epidemiology**

The relatively recent abundance of foods high in sugar, fat, and calories (Figure 9-2), as well as cigarette smoking and the overall adoption of an American lifestyle, contribute to the current health profile of the Native Americans. Today, chronic conditions account for six of the ten leading causes of death, and obesity and diabetes are affecting Indian communities at epidemic proportions (IHS 2005). The chronic conditions affecting the Potawatomi are correlated with obesity, but obesity may not be the sole explanation for the high rates of diabetes and related pathologies so common today. The reduction in physical activity due to motorized transportation, office jobs, video entertainment, and access to goods, services, and information through the Internet contributes to insulin resistance independently, as well as to obesity. Less



**FIGURE 9-2. Food vendors at a Prairie Band Potawatomi Pow-Wow, 2009. Foods being sold include typical fair and carnival foods such as funnel cakes, corn dogs, pork tenderloin, fried chicken fingers, Indian tacos, kettle corn, and curly fries.**

physical activity thus acts in two ways to contribute to metabolic syndrome, and changes in diet and overall lifestyle contribute to current disease profiles.

Many years and numerous political approaches to the “Indian problem” have failed to achieve Indian equality in health and welfare. Despite health improvements, a number of measures of health status indicate that Indian people continue to suffer from a variety of diseases at rates disproportionate to other racial and ethnic groups. Indian people still die at younger ages than other groups (Berry et al. 2004). Indian health status still lags behind all other U.S. racial and ethnic populations, and the health disparity gap between Indians and the general U.S. population is increasing (IHS 2005). Unlike the general population, in which rates of heart disease and cancer are declining, heart disease and cancer mortality among Indian people is still increasing. Diabetes death rates also continue to increase (Espey, Paisano, and Cobb 2005; Rhoades 2003). For the Prairie Band, trends for heart disease and diabetes deaths may be downward, but overall rates are still high, and the trend for cancers is increasing.

Prior to World War II, cardiovascular diseases were rare among Indians. Now heart disease is the leading cause of death. Strokes are the fifth leading cause of death today, and the rates for both of these cardiovascular diseases are on the rise. Modern cardiovascular disease rates among Indians are double that of the rest of the U.S. population. The dramatic increase of these rates is most likely due to high rates of diabetes, hypertension, and associated poor eating habits and physical inactivity (Berry et al. 2004). Native Americans in Jackson and Shawnee counties in particular show death rates from heart disease that are higher than in the larger Native American population.

In general Indians still have lower cancer incidence and mortality rates than other racial and ethnic groups, but as cancer rates decline among the general U.S. population, the rates are increasing among Indian people. Additionally, cancer survival rates among Indians are the lowest of any ethnic group in the U.S. (Berry et al. 2004; Espey, Paisano, and Cobb 2005; Wolsey and Cheek 1999). Among the Prairie Band, cancers have increased more dramatically than any other cause of death since 1974.

Deaths due to diabetes saw a dramatic increase between the two time-periods examined, reflecting the rising trend for diabetes in national data for Native Americans in the 1960s. Community elders, asked to recall when they first heard of diabetes, often reported it affecting an elderly parent, beginning in the 1940s and 1950s. Others recall not hearing about it until the 1980s. Diabetes was not among the top 10 causes of mortality at the time the IHS was founded, but over the last 50 years, deaths due to diabetes have quadrupled, making it the fourth leading cause of Indian mortality. Diabetic complications including heart disease, renal failure, blindness, and limb-loss have increased dramatically since 1955 (IHS 2005; Wolsey and Cheek 1999). Diabetes is now among the most serious health challenges facing Indian people,

contributing substantially to morbidity and mortality. Today, some Indian tribes have the highest rates of diabetes in the world, with over half of their adults suffering from the disease. Also significant is that heart disease deaths, which are the leading cause of deaths among Indians, are most often caused by diabetes. Thus, diabetes devastates Indian communities both in terms of quality of life and premature mortality. In addition to the toll taken in health and lives, the financial cost of diabetes is staggering. Forty percent of the total health care budget for Indians is for treating diabetes (Berry et al. 2004). While diabetes deaths among Indians in Jackson and Shawnee counties decreased slightly, the prevalence and incidence of the disease have been increasing, continuing to deplete health resources and impacting quality of life.

Some infectious diseases still show higher mortality rates among Indians than the general U.S. population. Sources report the age-adjusted death rate due to pneumonia and influenza among Indians is 67-71% higher than the rate for the U.S. population as a whole. Though tuberculosis among Indians has been on the decline for years, it is still more common among Indians than among the general U.S. population, causing four times as many deaths (Berry et al. 2004; IHS 2004; Wolsey and Cheek 1999). The death rate from the recent H1N1 influenza (swine flu) pandemic was four times greater in American Indians and Alaska Natives than in the rest of the U.S. population (Brown 2009). Infectious diseases have been on the decline among the Prairie Band community in the recent decades, however, and recent influenza and pneumonia death rates were slightly below those of U.S. whites.

Indian people also face a higher risk of mental health problems than do other racial or ethnic groups in the U.S. They are among the neediest groups for mental health care services year after year. The most common mental health problems and social pathologies among Indians today are depression, anxiety, interpersonal violence, suicides, and substance abuse (Berry et al.

2004). Alcoholism is seven times more common among Indian people than in the rest of the nation. Homicide rates are 1.5 times higher, and suicide rates are nearly double that of the general U.S. population (IHS 2005). Indians experience injuries at rates that range from 1.5 to 5 times that of the rest of the nation. The age-adjusted death rate for injuries among Indian people is 200 percent higher than the general population of the U.S. (Berry et al. 2004; IHS 2004). In contrast, Native Americans in Jackson and Shawnee counties have lower rates of suicide and homicide than Native Americans as a group, and have slightly lower rates of suicide than U.S. whites.

Though there have been recent improvements in infant mortality among Native Americans, disparities remain. Some sources report that Native American infants die 150% more often than white infants (Berry et al. 2004). Sudden infant death syndrome (SIDS) is three to four times more common among Indians than among whites. Native American infants are also much more likely to die from unintentional injuries and pneumonia and influenza than the general U.S. infant population (IHS 2004). Native Americans tend to have more of the associated risk factors for infant mortality, such as parental cigarette smoking, younger maternal age, and poverty, than whites (Irwin, Mannino, and Daling 1992; Scholer, Hickson, and Ray 1999; Sullivan and Barlow 2001).

The history of Native Americans in the U.S. is one of deprivation. U.S. population expansion, the reservation system, and allotment impoverished Indian people, disrupted traditional subsistence, and led to regular food shortages and nutritional inadequacies over several generations. Even as Native Americans modernized, they remained disadvantaged in economic opportunities, nutritional intake, living conditions, and health care services. Economic deprivation played a key role in the pattern of epidemiologic transition and current health

conditions. Poverty contributed to a lack of modern health care and living standards, delaying the epidemiologic transition among Native Americans. As Indians modernized they continued to be poor.

Poverty and unemployment are closely related to health disparities through reduced expenditures on health care, more social pathologies, and higher rates of alcohol consumption and cigarette smoking (Castor et al. 2006; Flint and Novotny 1997; Roubideaux 2002; Singh and Hoyert 2000; Trovato 1988). Also, income insecurity is associated with reduced expenditures on food, lower consumption of fruits and vegetables, and less nutritional diets (Drewnowski and Specter 2004). A prevailing condition for the Prairie Band community throughout much of the 20<sup>th</sup> century was relative poverty. Even as the Prairie Band improved their economic status, health disparities deriving from a history of impoverishment remained.

### *Transgenerational Effects*

Poverty may be seen as an inherited condition. Low socioeconomic status leads to earlier ages of pregnancy, inferior prenatal care, and resultant intrauterine fetal stress. Poverty is also associated with greater exposure to hazardous environmental neurotoxins. The poor rarely receive the level of health care as those who are more affluent. Psychosocial stress among the poor is more prevalent due to family dysfunction and violence exacerbated by economic struggles and psychiatric problems such as chronic stress and anxiety (Agin 2010). High levels of the stress hormone cortisol have been correlated with low-socioeconomic status, contributing to higher rates of metabolic and cardiovascular diseases (Cohen et al. 2006; Plat et al. 2006; Thayer and Kuzawa 2014). The consequences of exposure to stress during pregnancy include low-birthweight, negative postnatal infant disposition, and poor physical and mental development in childhood. Combined with earlier ages at pregnancy that reduce educational



opportunities and increase financial burdens, the cycle of poverty continues as the circumstances of poverty are passed from parents to children (Agin 2010; Thayer and Kuzawa 2014).

Persistent poverty reflects the trauma and disadvantages experienced by Native Americans over time. The U.S. Substance Abuse and Mental Health Service Administration (SAMHSA) describes trauma as resulting from events or circumstances experienced by individuals that are either physically or emotionally damaging. This trauma is seen as having long-term negative effects on an individual's well-being and physical, social, emotional, or spiritual welfare. SAMHSA further recognizes that communities as a whole can experience trauma, and that trauma can shape culture by being incorporated into cultural norms and so passed from one generation to the next (SAMHSA 2012).

Native American history following contact with Europeans is defined by trauma. The people and cultures suffered from heightened disease stress, warfare, genocide, loss of land, involuntary relocation, imposition of a new language and religion, and forced abandonment of traditional subsistence practices and foods. External conflict intensified internal conflict. Internal conflict and the loss of purpose contributed to social pathologies that were experienced or witnessed by most of the Native American society. Boarding schools separated families and forced Indian children into a foreign world where everything they knew was labeled as wrong. These circumstances bequeathed a heritage of trauma that still touches tribal communities (Brave Heart and DeBruyn 1998). What is more, the new understanding of the role of epigenes on health (Kuzawa and Sweet 2009; Kuzawa and Thayer 2011; Thayer and Kuzawa 2014) suggests that the legacy of trauma is passed down not only by culture, but in the actual biology of the victims. Thus structural violence affects the health of Native American groups such as the

Prairie Band by altering their biology through epigenetic means. Inequality becomes embodied in the physical health status of the people.

As suggested previously, the low-cost, high-fat, high-calorie foods most available to many Native Americans have direct consequences on health. However, their current dietary intake is only a part of the problem caused by the nutrition transition. The nutrition transition experienced by Native American populations such as the Prairie Band may be contributing to mortality from metabolic diseases in a more complicated manner than simple increases in sugars, fats, and calories. Research suggests that the experience of malnutrition in an individual can cause changes in gene expression that can be passed on to offspring and future generations. A study of a famine in the Netherlands uncovered a relationship between prenatal exposure to famine and insulin resistance in later life (Ravelli et al. 1998). Studying the effects of food surpluses and shortages on historical cohorts in Sweden, Kaati, Bygren, and Edvinsson (2002) and Pembry et al. (2006) discovered that parental and grandparental food supply mediated the development of diabetes and other chronic conditions in subjects, dependent upon genomic imprinting. Such studies suggest that the nutrition transition experienced by Native Americans may factor into current disease patterns, also through epigenetic means.

## **Summary and Closing**

The Prairie Band Potawatomi experienced the epidemiologic transition much as have other Native American groups; that is, a delayed entrance into the *Age of Degenerative and Man-Made Diseases*; a steep rise in the prevalence of certain chronic diseases to rates at or above those typical of the general U.S. population, particularly heart disease, diabetes, and chronic liver disease; and a high rate of deaths due to accidents. The native people of Jackson and Shawnee counties in Kansas differed from other Native American groups only in having higher death rates

due to heart disease and cancer and lower death rates for those causes related to social pathologies—accidents, suicides, and homicides.

Broudy and May (1983) concluded that by the 1980s the Navajo had passed through the *Age of Pestilence and Famine* and were in the latter stages of the *Age of Receding Pandemics*, somewhat straddling Stages Two and Three as described by Omran. The dramatic rise in behavior-related deaths was seen as additional evidence of an epidemiologic transition among the Navajo, albeit one with its own particular characteristics. The health care system was imposed upon the Navajo from the outside; thus, its effects were independent of the other factors associated with modernization. Infectious diseases were easily cured pharmacologically and were quickly eliminated as major causes of mortality, whereas fertility decreased slowly, leading to a younger population. This pattern is more common when health care methods and technology are introduced from the outside. Among the Navajo, behaviorally influenced conditions and deaths increased more rapidly with modernization than did chronic diseases, leading to a pattern of epidemiologic transition that differed markedly from that of the greater U.S. population (Broudy and May 1983). The Prairie Band community experienced similar shifts, with pharmacological and hygienic interventions for infectious diseases, a rise in behavioral and social pathologies leading to more accidents and alcoholic liver disorders, a nutritional transition, and a delayed rise in chronic diseases.

Considering the effects of structural violence and discrimination against Native Americans, it is difficult to conclude that genetic ancestry at the population level has a major impact on the expression of metabolic diseases. Genetic changes cannot explain the rapid rise and current epidemic of diabetes among Native Americans and other populations throughout the world. There simply has not been an adequate amount of time for changes in gene frequencies

alone to explain the increase in diabetes and obesity rates over just the last few generations (Kunitz 2004; Lappe 1994; Szathmary 1994; Yach, Stuckler, and Brownwell 2006; Young 1994).

Thrifty genes do not provide as satisfactory an explanation for the rise of obesity and metabolic diseases as other factors. Although the explanation for the diabetes epidemic among Native Americans most often implicates a genetic predisposition, the rise of type 2 diabetes directly results from the rapid social changes experienced by Indian people over the last 50 years. It reflects a political and economic situation that has been experienced among Native Americans and other indigenous people throughout the world. Diabetes is a byproduct of the U.S. federal government's systematic disruption of Indian diet and culture. Like suicide, alcoholism, substance abuse, and other social pathologies that afflict Indian people, diabetes is a colonial disease with social and economic causes that determine its inception and frequency (Campbell 1989). These conditions represent the modern diseases of poverty.

Of what value then are the thrifty genotype hypothesis and New World syndrome in explaining the recent rise of metabolic conditions among Native Americans? They may explain the early onset of the obesity and diabetes epidemics as compared to other populations. However, social and economic aspects also factor into the pattern of diabetes experienced by Native American populations. The original authors of both hypotheses recognized the interplay between genes and the environment. Acknowledging greater roles for other factors does not entirely negate the input of the thrifty gene. Yet, after decades of research, only a few genes have been identified that impact obesity, diabetes, and metabolic syndrome. Furthermore, these genes have not been shown to be unequivocal adaptations to periodic famines. New challenges to the hypothesis take greater account of more recent environmental conditions and our better

understanding of genetics. These new ideas make obesity and diabetes etiology more complex, but also empower individuals and society with a means to prevent the conditions through action.

The cause of variation in obesity and diabetes rates is an important scientific and scholarly question, but the thrifty gene hypothesis has seeped into the consciousness of the average American. Indeed, many Native Americans are able to describe the New World syndrome as the cause of diabetes among their people. In this regard, that component of structural violence in which the hegemonic power is able to impose its views on those who are oppressed—such as when Native Americans themselves explain their susceptibility to diabetes as something inherent in their own biological makeup—has been effective. As understood by most laypeople, the thrifty gene and New World syndrome are simplistic explanations employing biological determinism, with colonialism intertwined as a part of the inevitability of the condition among Native Americans. For the Native American public, greater emphasis should be placed on prevention by addressing those factors shown to impact obesity and diabetes to a greater degree – diet, exercise, pre- and post-natal care, and economic circumstances. Research has failed to adequately support either the thrifty genotype hypothesis or New World syndrome as the explanations for variation in obesity and diabetes among individuals or populations. On the contrary, recent research has called into question the basic assumptions of these concepts, suggesting that the etiology of these metabolic conditions is more complex, involving an interaction of genetic, epigenetic, developmental, environmental, societal, and economic factors. Considering the influence socioeconomic factors appear to have on health, it is unlikely that population-wide genetic changes resulting from historic or prehistoric adaptations contribute substantially to the Native American-specific pattern of epidemiologic transition.

In 1909, physical anthropologist Aleš Hrdlička suggested that Native American susceptibility to tuberculosis was biologically based, an assumption that persisted during the racially charged anti-immigrant context of early 20<sup>th</sup> century America. This perspective began to be challenged in the 1920s, however, and research intensified in the 1930s. By the 1950s, the environment was the predominant explanation for high rates of TB among Native Americans (McMillen 2008). In 1923 the Committee of the National Tuberculosis Association reported:

Those who believe that tuberculosis attacks without any racial preferences, but that the ravages of the disease are greatly influenced by unfavorable surroundings and economic conditions, and that the advent of the white man created a struggle for existence and subsistence which favored the development of the disease and that the red man, like the white or black races, responds to improved environments, will find in the following pages considerable evidence in support of this opinion (Kober et al. 1923:4).

The thrifty genotype hypothesis, as a biological explanation for diabetes and obesity among Native Americans, serves the same purpose as biological explanations did for TB 100 years ago. That is that the disease primarily results from constitutional makeup, with indigenous peoples viewed as being less genetically fit for civilization than their Old World counterparts. However, it seems more likely that the environment in which Native Americans find themselves – featuring social and economic disparities arising from structural violence that lead to inadequate diet and health care – is the primary culprit in the disparity of diabetes prevalence, as it was with tuberculosis in the past. Additionally, epigenetic changes initiated by socioeconomic conditions may have contributed to the illusion of genetic predisposition.

Diabetes is not just a disease of Native Americans. Diabetes and obesity rates throughout the rest of the country and the world are also rising (IDF 2011). Native Americans were impacted more quickly and severely by the rise of the diabetes epidemic due to environmental circumstances. Non-Native American populations were buffered by conditions such as access to a higher-quality diet and better preventive health care services. The nation and world are taking notice of diabetes as an epidemic some 30 years after it became entrenched among Native Americans.

Health disparities remain despite progress in general Indian health. The current health disparities are primarily a social problem. These disparities reflect years of mistreatment and tribal suspicion of the federal government that have prompted many tribes to reject federal programs offering positive changes in policy, such as the Indian Reorganization Act. Termination was an attempt to eliminate the special status of Indian people altogether, and many tribes subsequently suffered the loss of funding and services. Self-determination has not always been as straightforward as its name suggests. Despite transferring some control of programs to tribes, determining appropriations has ultimately remained with the federal government. Inconsistent and inadequate funding undermined self-determination and impacted the ability of tribes to develop programs most beneficial to their members. This is a primary feature of the structural violence affecting Native Americans. Regardless of feelings that the U.S. government is obligated to provide health care services to Native Americans, tribes such as the Prairie Band Potawatomi will likely have to bear a greater burden of the financial responsibility for providing health care to tribal members, particularly if the health services and status are to reach levels comparable to other segments of the U.S. society.

The Prairie Band Potawatomi have had some success in improving the economic conditions of members of the tribe, primarily through their casino revenue. Tribal members are candid regarding the improvements in resources and services available to them. In an attempt to improve health services to their members, in 2004 the Potawatomi assumed control of their health clinic from the IHS under the compacting/contracting rules of the Indian Self-Determination Act. The tribal leadership then contracted with an outside firm, CRAssociates, to manage their clinic. At the time, the clinic was located off the reservation, nine miles to the north in the town of Holton. In 2007 the tribe completed construction of a new \$7 million health center located on the reservation, paid for primarily with casino revenue. The new clinic provided better access for patients living on the reservation and in Topeka. In April 2009 the tribe took over direct management of their clinic from CRAssociates.

As indicated by Cunningham (1996), the finite IHS budget often forced tribal clinics, such as the Prairie Band Potawatomi clinic, to adjust the contract services it could cover. As funds for a fiscal year were depleted, services that were at a lower priority level were simply not paid for, leaving patients with a bill or causing them to opt against seeking those services. Native Americans often choose not to pay out-of-pocket for medical services not covered by IHS funds (Perrott and West 1957): Potawatomi patients often refused preventive procedures not covered by the clinic. Such situations lead to delays in diagnosing conditions and the development of more acute conditions.

As a result of IHS funding constraints, the Prairie Band Potawatomi clinic has regularly adjusted which contract service tests and procedures it would remit, based on the severity of the condition and need for the procedure. Adjustments followed the IHS guidelines for priorities of care for contracted health services (IHS 1995). The IHS medical priorities levels are:



- I      Emergent/Acutely Urgent Care Services
- II     Acute Primary and Preventive Care Services
- III    Chronic Primary and Secondary Care Services
- IV    Chronic Tertiary Care Services
- V     Excluded Services

As funds for a given fiscal year were depleted, services at a lower level were no longer covered, leading patients to choose not to undergo diagnostic tests or receive preventive services.

Available funding also affected the formulary of the clinic's pharmacy. With a limited budget and little or no remittance from patients, the pharmacy had to limit the drugs it kept available to those that were less expensive and often older, making it difficult for some patients to take advantage of new drug developments and more expensive treatments.

Those most likely to utilize tribal clinic services funded by IHS were more likely to be poor or have no additional health insurance, since the direct services of the tribal clinic were free. Many tribal members were reluctant to talk openly about their incomes in an official capacity. Often this reflects a fear that services will be reduced or withheld if their income exceeds a certain threshold. This reluctance became an issue when the tribal clinic began to require patients without private or employer-supplied insurance to apply for Medicaid eligibility. Tribal clinics struggle to get patient cooperation in applying for Medicare or Medicaid services for which they might qualify, which would allow the clinics to receive reimbursements for provided medical and pharmaceutical services. The results of this reluctance are delayed or cancelled screenings and treatments that otherwise could prevent more serious conditions leading to life-threatening illnesses; and fewer resources with which tribal clinics can provide services. Given

the funding scheme of the IHS, it is possible that a health disparity will develop within the Native American communities between those who rely solely on the IHS and those with supplemental insurance or additional tribal assistance.

Providing more resources for preventive care may prove to be a more cost-effective approach to reducing health problems among Native Americans. More funding to IHS and to tribally operated health facilities would provide for better care by allowing patients to receive services that might not otherwise be covered. Among the Prairie Band this care comes from outside providers through contracted services, requiring more funds to pay for these services.

Indian people are entitled to health care as a result of treaty negotiations (McCabe 1999). Many Indian people believe their health status and lives will improve once the government meets those treaty obligations. But true self-determination for Indian people may come only through greater self-sufficiency. With the failure of the U.S. government to live up to its trust and treaty responsibilities, Native Americans must be more reliant upon their tribal governments to provide health care services, particularly those tribes that have resources. Currently, the quality of health care for Indian people is still primarily dependent on federal policies and federal dollars. Tribes will most likely find funding and services to be inadequate for as long as they are provided by a conflicted government. Sustainable economic development may allow for tribes themselves to provide the resources necessary to close the disparity gap between Indians and the rest of America. Such economic development is ultimately dependent upon tribal governments that affirm and act on the collective interests of the tribe.

Structural violence continues to affect Native American health through inadequate funding of health services and a social system that perpetuates poverty in native communities. Structural violence has a long legacy of harm against Native Americans that will continue for

years to come as living tribal members experience health care and nutritional inadequacies with biological effects that can be passed on to subsequent generations. Ending health disparities among Native Americans will require adequate funding for a health care program that allows for more preventive services and comprehensive prenatal care. More and better economic opportunities will give Native Americans the resources to afford better health care and to purchase and prepare healthier foods. Greater economic opportunities should also reduce the burden of social pathologies so prevalent among native peoples.

The mortality pattern exhibited by Native Americans in Jackson and Shawnee counties is indicative of disparities compared to the overall U.S. population pattern, reflecting historical poverty and continued economic hardships. Fewer deaths from social pathologies compared to the rates for all Native Americans suggests that Native Americans of the Prairie Band community have a better quality of life than some other tribes, with lower levels of infectious diseases and fewer deaths from social pathologies. Gambling revenues provide the tribe with resources for more services and jobs for tribal members, perhaps buffering many of the Native Americans in Jackson and Shawnee counties from these conditions. The increase in income, however, also provides more resources to purchase high-fat, high-calorie fast food and restaurant foods, contributing to obesity and the diseases associated with the condition.

### **Further Research**

Long-term epidemiological studies of a tribe's health are challenging. First, tribal leadership changes often. With political changes often come changes in attitudes toward research. Studies deemed appropriate and useful at one time by one council may be discontinued altogether by a succeeding council. Suspicious of outside researchers asking probing questions or requesting access to private information, tribal members can raise their concerns at tribal

member meetings (General Council) or to the Tribal Council, making support of research studies a political liability. Secondly, few tribal leaders or tribal members recognize the value of socioeconomic data in studying health. Income in particular is a sensitive subject on which little cooperation is provided. People with a lay understanding of diabetes as being determined by genes and diet are skeptical of diabetes research studies that ask for seemingly unconnected data about income, educational attainment, mental health, ancestry, and ancestral living conditions.

Given the present political makeup and views of the current Prairie Band tribal leadership, more extensive research will have to be initiated internally or by a member of the tribe. Such an effort requires that the tribal leadership recognize the value of wide-ranging data and how such data can be used to formulate strategies to provide more and better services. A community based participatory research approach may have success and support in the community, but without trust and understanding of research, the tribe is less likely to initiate research projects with outside assistance. Areas for further research are suggested below.

Individual incomes should be quantified to determine what impact economic status is having on health. Examination of educational attainment should be done for the same purpose. It also would be useful to know who exactly is utilizing the tribal services; whether there are any differences in health status related to tribal clinic utilization; and whether income level is a factor in tribal clinic utilization. This might provide a measure of the effectiveness of the IHS contracted services compared to those provided through other means. Contract support payments by IHS are currently a contentious issue between the tribes and IHS. Quantifying the health effects of contract payment shortfalls could provide tribes with further evidence of the government's failure to live up to its treaty obligations.

A genetic analysis of the Prairie Band and other Native American groups for the gene frequencies of those traits thus far identified as contributing to metabolic syndrome and related conditions would provide more evidence for evaluating the validity of the New World syndrome. Of particular interest would be a Native American-specific analysis of the genetics of fat patterning, given the known relationship between body fat patterning and risk for metabolic diseases. Should Native American populations demonstrate higher rates for the gene variants contributing to metabolic diseases, perhaps a case for the New World syndrome can continue to be made. Making a connection between the gene variants and the particular prehistoric circumstances of Native American populations would be the next challenge.

Epigenetics is a promising area of research for the biological tendency for metabolic syndrome. Historical trauma of Native Americans in previous generations is well documented. Transgenerational epigenetics provides a mechanism through which to consider intergenerational trauma among Native Americans. A study similar to Pembry et al.'s (2006) study in Sweden of the transgenerational transmission of diabetes risk to grandchildren based on grandparents' food supply would be of interest. Such a study might be possible using the original 1928 health survey, which identified those surveyed by name, household, and birth date when available. Those data could be used in conjunction with clinic data on metabolic conditions of current patients, connecting ancestors and descendants through genealogy.

Future studies such as those suggested could better elucidate the manner in which health disparities persist in communities like that of the Prairie Band. Currently, efforts are being made by the Indian tribes in Kansas to get mortality data for their communities. It is anticipated that the data will be used to develop strategies for more effectively addressing health disparities. As the tribal leaders become more aware of the various underlying factors that contribute to

variations in health and disease, it is likely that the tribes will endeavor to include socioeconomic data as a means to understanding the dynamics of health.

## Chapter 9 References Cited

Agin, D. 2010. *More Than Genes: What Science Can Tell Us About Toxic Chemicals, Development, and the Risk to Our Children*. New York: Oxford University Press.

Berry, Mary F., Reynoso, C., Braceras, J.C., Edley, Jr., Kirsanow, P.N., Meeks, E.M., Redenbaugh, R.G., and Thernstrom, A. 2004. *Broken Promises: Evaluating the Native American Health Care System*. U.S. Commission on Civil Rights. Washington, D.C.: U.S. Government Printing Office.

Brave Heart, M. and DeBruyn, L.M. 1998. The American Indian Holocaust: Healing Historical Unresolved Grief. *American Indian and Alaska Native Mental Health Research* 8:60-82.

Broudy, D.W. and May P.A. 1983. Demographic and Epidemiologic Transition among the Navajo Indians. *Social Biology* 30(1):1-16.

Brown, David 2009. *H1N1 Death Rate is Elevated for Indians, Alaska Natives*. The Washington Post, December 11, 2009.

Brownson, R.C. and Boehmer, T.K. 2004. "Patterns and Trends in Physical Activity, Occupation, Transportation, Land Use, and Sedentary Behaviors," Commissioned Paper for TRB Special Report 282 *Does the Built Environment Influence Physical Activity? Examining the Evidence*. Washington, DC: Transportation Research Board.

Campbell, G.R. 1989. The Changing Dimension of Native American Health: A Critical Understanding of Contemporary Native American Health Issues. *American Indian Culture and Research Journal* 13(3-4):1-20.

Carter, S.B. and Sutch, R. 2006. "American Indians," in *Historical Statistics of the United States, Millennial Edition*. Edited by S. Carter, S. Gartner, M. Haines, A. Olmstead, R. Sutch, and G. Wright, pp. 715-725. New York: Cambridge University Press.

Castor, Mei L., Smyser, Michael S., Taulii, Maile M., Park, Alice N., Lawson, Shelley A., and Forquera, Ralph A. 2006. A Nationwide Population-Based Study Identifying Health Disparities Between American Indians/Alaska Natives and the General Populations Living in Select Urban Counties. *American Journal of Public Health* 96:1478-1484.

CDC (Centers for Disease Control and Prevention). 2003. Surveillance for Health Behaviors of American Indians and Alaska Natives, Findings from the Behavioral Risk Factor Surveillance System, 1997 - 2000. *Morbidity and Mortality Weekly Report* 52(SS-7).

Cohen, S., Schwartz, J. E., Epel, E., Kirschbaum, C., Sidney, S., and Seeman, T. 2006. Socioeconomic Status, Race, and Diurnal Cortisol Decline in the Coronary Artery Risk Development in Young Adults (CARDIA) Study. *Psychosomatic Medicine* 68(1):41-50.

- Cunningham, P. J. 1996. "Health Care Utilization, Expenditures, and Insurance Coverage for American Indians and Alaska Natives Eligible for the Indian Health Service," in *Changing Numbers, Changing Needs: American Indian Demography and Public Health*. Edited by Gary D. Sandefur, Ronald R. Rindfuss, and Barney Cohen, pp. 289-314. Committee on Population, Commission on Behavioral and Social Sciences and Education, National Research Council. Washington, D.C.: National Academy Press.
- Drewnowski, A. and Spector, S.E. 2004. Poverty and Obesity: The Role of Energy Density and Energy Costs. *American Journal of Clinical Nutrition* 79:6-16.
- Edmunds, R.D. 1978. *The Potawatomis: Keepers of the Fire*. Norman, OK: University of Oklahoma Press.
- Espey, D., Paisano, R. and Cobb, N. 2005 Regional Patterns and Trends in Cancer Mortality among American Indian and Alaska Natives, 1990–2001. *Cancer* 103:1045-1053.
- Flint, A.J. and Novotny, T.E. 1997. Poverty Status and Cigarette Smoking Prevalence and Cessation in the United States, 1983-1993: The Independent Risk of Being Poor. *Tobacco Control* 6:14-18.
- French, M. 1997. *U.S. Economic History Since 1945*. New York: Manchester University Press.
- Hill Jr., C.A. and Spector, M.I. 1971. Natality and Mortality of American Indians Compared with U.S. Whites and Nonwhites. *HSMHA Health Reports* 86(3):229-246.
- Howell, N.A. 1970. *Potawatomi Pregnancy and Childbirth*. Master's Thesis, The University of Kansas.
- Hrdlička, Aleš 1909. *Tuberculosis among Certain Indian Tribes of the United States*. Smithsonian Institution, Bureau of American Ethnology, Bulletin 42. Washington, DC: U.S. Government Printing Office.
- IDF (International Diabetes Federation). 2011. One Adult in Ten had Diabetes in North America. IDF North American Press Release, <http://www.idf.org/sites/default/files/attachments/NAC-Press-Release-WDD.pdf>. November 14<sup>th</sup>, 2011.
- IHS (Indian Health Service) 1974. *Indian Health Trends and Services, 1974 Edition*. Washington, DC: U.S. Department of Health, Education, and Welfare.
- . 1995. Contract Health Services, Requirements - Priorities of Care. [http://www.ihs.gov/nonmedicalprograms/chs/index.cfm?module=chs\\_requirements\\_priorities\\_of\\_care#2](http://www.ihs.gov/nonmedicalprograms/chs/index.cfm?module=chs_requirements_priorities_of_care#2). October 25<sup>th</sup>, 2011.
- . 1995. Contract Health Services, Requirements - Priorities of Care. [http://www.ihs.gov/nonmedicalprograms/chs/index.cfm?module=chs\\_requirements\\_priorities\\_of\\_care#2](http://www.ihs.gov/nonmedicalprograms/chs/index.cfm?module=chs_requirements_priorities_of_care#2). October 25<sup>th</sup>, 2011.
- . 2004. *Trends in Indian Health, 2000-2001*. Rockville, Md.: U.S. Department of Health and Human Services.
- . 2005. *The First 50 Years of the Indian Health Service: Caring and Curing*. Rockville, Md.: U.S. Department of Health and Human Services.

Irwin, K.L., Mannino, S., and Daling, J. 1992. Sudden Infant Death Syndrome in Washington State: Why are Native American Infants at Greater Risk than White Infants? *Journal of Pediatrics* 121(2):242-247.

Kaati, G., Bygren, L.O., and Edvinsson, S. 2002. Cardiovascular and Diabetes Mortality Determined by Nutrition During Parents' and Grandparents Slow Growth Period. *European Journal of Human Genetics* 10:682-688.

Kelly, C.G. 1952. *Welfare Aspects of the Potawatomi Indian Agency*. Master's Thesis, The University of Kansas.

Kober, G.M., Baldwin, W.H., Dearholt, H.E., Tonkin, A.B., Bushnell, G.E., and Murphy, J.A. 1923. *Tuberculosis among the North American Indians*. Report of a Committee of the National Tuberculosis Association. Washington, D.C.: U.S. Government Printing Office.

Kunitz, S.J. 2004. "The Evolution of Disease and the Devolution of Health Care for American Indians," in *The Changing Face of Disease: Implications for Society*. Edited by N. Mascie-Taylor, J. Peters, and S. McGarvey, pp. 153-169. Society for the Study of Human Biology Series: 43. Boca Raton, FL: Routledge.

Kuzawa, C.W. and Sweet, E. 2009. Epigenetics and the Embodiment of Race: Developmental Origins of US Racial Disparities in Cardiovascular Health. *American Journal of Human Biology* 21:2-15.

Kuzawa, C.W. and Thayer, Z.M. 2011. Timescales of Human Adaptation: The Role of Epigenetic Processes. *Epigenomics* 3(2):221-234.

Lappe, M. 1994. *Evolutionary Medicine: Rethinking the Origins of Disease*. San Francisco: Sierra Club Books.

McCabe, M. 1999. "Health Care of American Indian and Alaska Native Elders," in *Primary Care of Native American Patients: Diagnosis, Therapy, and Epidemiology*. Edited by J.M. Galloway, B.W. Goldberg, and J.S. Alpert, pp. 323-329. Boston: Butterworth Heinemann.

McElvaine, Robert S. 1984. *The Great Depression*. New York: Times Books.

McMillen, C.W. 2008. The Red Man and the White Plague: Rethinking Race, Tuberculosis, and American Indians, ca. 1890-1950. *Bulletin of the History of Medicine* 82(3):608-645.

Neel, J.V., Weder, A.B., and Julius, S. 1998. Type II Diabetes, Essential Hypertension, and Obesity as "Syndromes of Impaired Genetic Homeostasis": the "Thrifty Genotype" Hypothesis Enters the 21st Century. *Perspectives in Biology and Medicine* 42(1):44-74.

Omran, A.R. 1971. The Epidemiologic Transition; A Theory of the Epidemiology of Population Change. *The Milbank Memorial Fund Quarterly* 49(4):509-538.

Pembrey, M.E., Bygren, L.O., Kaati, G., Edvinsson, S., Northstone, K., Sjöström, M., Golding, J., and the ALSPAC Team. 2006. Sex-Specific, Male-Line Transgenerational Responses in Humans. *European Journal of Human Genetics* 14:159-166.



- Perrott, George St. J. and West, M.D. 1957. Health Services for American Indians. *Public Health Reports (1896-1970)* 72(7): 565-570.
- Popkin, B.M. 1993. Nutrition Patterns and Transitions. *Population and Development Review* 19:138-157.
- Putnam, R.D. 1995. Tuning In, Tuning Out: The Strange Disappearance of Social Capital in America. *Political Science and Politics* 28(4):664-683.
- Putney, Diane T. 1980. *Fighting the Scourge: American Indian Morbidity and Federal Policy, 1897 – 1928*. PhD Dissertation. Milwaukee, WI: Marquette University.
- Ravelli, A.C.J., van der Meulen, J.H.P., Michels, R.P.J., Osmond, C., Barker, D.J.P., Hales, C.N., and Bleker, O.P. 1998. Glucose Tolerance in Adults after Prenatal Exposure to Famine. *Lancet* 351:173-177.
- Rhoades, Everett R. 2003. The Health Status of American Indian and Alaska Native Males. *American Journal of Public Health* 93:774-778.
- Roubideaux, Y. 2002. Perspectives on American Indian Health. *American Journal of Public Health* 92:1401-1403.
- SAMHSA (Substance Abuse and Mental Health Services Administration). 2012. Trauma Definition. <http://www.samhsa.gov/traumajustice/traumadefinition/definition.aspx> October 10th, 2012.
- Scholer, S.J., Hickson, G.B., and Ray, W.A. 1999. Sociodemographic Factors Identify US Infants at High Risk of Injury Mortality. *Pediatrics* 103(6):1183-1188.
- Searcy, Ann M. 1965. *Contemporary and Traditional Prairie Potawatomi Child Life*. KU Potawatomi Study Research Report Number 7. Lawrence, Kansas: Department of Anthropology.
- Sells, C. 1919. *Annual Reports of the Commissioners of Indian Affairs*. Office of Indian Affairs. Washington, D.C.: U.S. Government Printing Office.
- Singh, G.K., and Hoyert, D.L. 2000. Social Epidemiology of Chronic Liver Disease and Cirrhosis Mortality in the United States, 1935 – 1997: Trends and Differentials by Ethnicity, Socioeconomic Status, and Alcohol Consumption. *Human Biology* 72(5):801-820.
- Sullivan, F.M., and Barlow, S.M. 2001. Review of Risk Factors for Sudden Infant Death Syndrome. *Paediatric Perinatal Epidemiology* 15(2):144–200.
- Szathmary, E.J. 1994. “Factors that Influence the Onset of Diabetes in Dogrib Indians of the Canadian Northwest Territories,” in *Diabetes as a Disease of Civilization: The Impact of Culture Change on Indigenous Peoples*. Edited by J. Joe and R. Young, pp. 229-268. New York: Mouton de Gruyter.
- Thayer, Z.M. and Kuzawa, C.W. 2014. Early Origins of Health Disparities: Material Deprivation Predicts Maternal Evening Cortisol in Pregnancy and Offspring Cortisol Reactivity in the First Few Weeks of Life. *American Journal of Human Biology* DOI: 10.1002/ajhb.22532.
- Townsend, J.G. 1938. Disease and the Indian. *The Scientific Monthly* 47(6):479-495.

Trovato, F. 1988. Mortality Differentials in Canada, 1951-1971: French, British, and Indians. *Culture, Medicine and Psychiatry* 12:459-477.

Wolsey, Darcy H., and Cheek, James, E. 1999. "Epidemiologic Patterns of Morbidity and Mortality," in *Primary Care of Native American Patients: Diagnosis, Therapy, and Epidemiology*. Edited by J.M. Galloway, B.W. Goldberg, and J.S. Alpert, pp. 7-16. Boston: Butterworth Heinemann.

Yach, D., Stuckler, D., and Brownwell, K.D. 2006. Epidemiologic and Economic Consequences of the Global Epidemics of Obesity and Diabetes. *Nature Medicine* 12(1):62-66.

Young, T.K. 1994. *The Health of Native Americans: Toward a Biocultural Epidemiology*. Oxford: Oxford University Press.

—. 1996. "Recent Health Trends in the Native American Population," *Changing Numbers, Changing Needs: American Indian Demography and Public Health*. Edited by G. Sandefur and B. Cohen, pp. 53-75. Washington, D.C.: National Academy Press.

\*Also published in *Population Research and Policy Review* 1997. 16:146-147.

# APPENDIX A

## Potawatomi Health Survey Form, 1928

STUDY OF HEALTH OF INDIANS - POTTAWATOMIE RESERVATION, MAYETTA, KANS.

I. FAMILY NAME \_\_\_\_\_ ADDRESS \_\_\_\_\_

1. Father's Name \_\_\_\_\_ Living? \_\_\_\_\_ Dead? \_\_\_\_\_

2. Mother's Name \_\_\_\_\_ Living? \_\_\_\_\_ Dead? \_\_\_\_\_

II PERCENT OF INDIAN BLOOD \_\_\_\_\_ TRIBE? \_\_\_\_\_

III MEMBERS OF HOUSEHOLD:

(Adults)

(Age)

(Children)

(Age)

_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____

IV. OCCUPATION (Head of Family) \_\_\_\_\_

V. OCCUPATION OF OTHER WAGE EARNERS \_\_\_\_\_

VI. NO. IN FAMILY ABLE TO READ AND WRITE: \_\_\_\_\_

VII TOTAL ANNUAL INCOME OF FAMILY: \_\_\_\_\_

VIII HOUSING CONDITIONS:

1. Size of House? \_\_\_\_\_ 3. Sleeping Quarters: \_\_\_\_\_

2. Sanitary Condition: \_\_\_\_\_ a. No. to room? \_\_\_\_\_

b. Fair? \_\_\_\_\_ b. Ventilation? \_\_\_\_\_

c. Poor? \_\_\_\_\_ 4. Individual Towels, etc.,

used? \_\_\_\_\_

IX EXCRETA DISPOSAL:

1. Outside privies? \_\_\_\_\_ Pits? \_\_\_\_\_ Protected from flies? \_\_\_\_\_

2. Absent from soil pollution? \_\_\_\_\_ Remarks: \_\_\_\_\_

WATER SUPPLY.

1. Well? \_\_\_\_\_ Spring? \_\_\_\_\_ Cistern? \_\_\_\_\_ Tested? \_\_\_\_\_

2. Protected from contamination? \_\_\_\_\_

3. Remarks: \_\_\_\_\_

# **APPENDIX A** **Potawatomi Health Survey Form, 1928**

2

**XI FAMILY HISTORY** (Age and cause of death of other members of family)  
 (Name) (Age) (Cause of Death)

_____	_____	_____
_____	_____	_____
_____	_____	_____
_____	_____	_____
_____	_____	_____

**XII. HEALTH CONDITION OF THE LIVING MEMBERS:**

Name	Condition:	Single, Married Widowed or Divorced:
_____	_____	_____
_____	_____	_____
_____	_____	_____
_____	_____	_____
_____	_____	_____
_____	_____	_____

**XIII SERIOUS ILLNESS IN PAST FIVE YEARS:**

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

**XIV. TUBERCULOSIS IN FAMILY:** \_\_\_\_\_

**XV. MEDICAL SERVICE?** \_\_\_\_\_

**XVI. DENTAL SERVICE?** \_\_\_\_\_ **NURSING SERVICE?** \_\_\_\_\_

\_\_\_\_\_ **MIDWIFERY?** \_\_\_\_\_

**XVII. DAILY MENU:** \_\_\_\_\_

\_\_\_\_\_

## **APPENDIX B**

### **DEFINITION OF THE “PRAIRIE BAND COMMUNITY”**

Defining a tribal group through time can be challenging; the legal criteria for tribal membership change over time, as do the cultural rules for determining tribal membership. Originally, tribal membership was determined culturally, based on aspects such as kinship patterns, marriage rules, locality, and/or clans. Allotment required lists of tribal members for land severalty, and these tribal “rolls” formed the legal basis of membership for many tribes.

The modern Prairie Band Potawatomi (PBP) of Kansas developed from the United Bands that settled first in Missouri, and then Iowa (Clifton 1998). These were the Potawatomi of southern Wisconsin and northern Illinois, also referred to as the Potawatomi of the Prairie and Kankakee, and included members of the Odawa and Ojibwa tribes (Edmunds 1978). Among the most influential leaders of the Potawatomi during the first half of the 19<sup>th</sup> century was Shabbona, who was actually an Odawa who had married into the Potawatomi. Today, the PBP claim land in Illinois given to Shabbona and are attempting to place the land in trust based on his role with the tribe.

The various “Potawatomi Agencies” provided the earliest information on the health conditions of the tribe after relocation to Kansas. However, these various incarnations of the agency were often responsible for multiple Indian tribes at different times, and did not always indicate tribal affiliation in reports on health and living conditions. At times, the agency responsible for Potawatomi affairs was also responsible for the Konza, Kickapoo, Chippewa, Munsee, Ioway, Sauk, and Fox tribes (Hill, 1981; NARA 2009). The 1928 health survey did record the tribe of each household. Approximately 78% were listed as Potawatomi exclusively.

The 1932 Baldwin Constitution of the PBP failed to specifically define tribal membership. A new constitution in 1961 defined tribal members as being anyone who had received allotments in accordance with the Dawes Act and their descendants. Degree of Indian

## **APPENDIX B**

### **DEFINITION OF THE “PRAIRIE BAND COMMUNITY”**

blood was not a requirement (Clifton 1998). An amendment to the constitution in 2000 made it necessary for tribal members to possess at least 1/4 PBP Nation blood (Prairie Band Potawatomi Nation 2011).

For several decades at least, the community on and around the PBP Reservation has included many Native Americans from other tribes. In particular, the Prairie Band have had a long and close relationship with the Kansas Kickapoo (Clifton 1969), and many of the followers of the Kickapoo Prophet, Kennekuk, were in fact, Potawatomi (Herring 1988). By the mid-1960s, well before the changes in enrollment, “exclusive” Prairie Band households only constituted 30% of the households on the reservation. Prairie Band and Citizen Potawatomi households constituted 19%; and Prairie Band and Kickapoo households made up an additional 11%. Households consisting of Prairie Band and members of other (non-Kickapoo) tribes were 19% of the reservation total. The remaining 21% consisted of Prairie Band members living with non-Natives (Clifton and Isaac 1964).

Poverty on rural reservations and the relocation policy during the termination period caused a demographic shift among tribes such as the Prairie Band. Only about 16% of Indians were living in urban areas in 1950. In contrast, by 2000, 60% of Indian people were living in urban areas (IHS 2005). Topeka is the closest urban center to the Prairie Band Reservation.

The KDHE Office of Vital Statistics data used for the fourth quarter of the 20<sup>th</sup> century did not include information on tribal affinity, so all records for Native Americans from Jackson and Shawnee counties were used. The bulk (~83%) of Prairie Band Potawatomi Health Center patients that were members of the PBP tribe lived in either Jackson (~60%) or Shawnee (~23%) counties. The next county with the highest percent was Brown, with only about 6%. Since Brown County is also the home of the Kickapoo Reservation and parts of the Sac & Fox and

## **APPENDIX B**

### **DEFINITION OF THE “PRAIRIE BAND COMMUNITY”**

Ioway Reservations, Brown County deaths were not included in the comparison with historical data identified as PBP. Topeka, in Shawnee County, had the most PBP tribal members of any city except Mayetta.

Given the known historical intermingling of the PBP with other tribes as described above, it was presumed that the early 20<sup>th</sup> century data included mostly PBP tribal members, but also a substantial number of non-PBP. Not having tribal affiliation in the data used for the last quarter of the 20th century, using data from both Jackson and Shawnee counties would provide more robust numbers for analysis while maintaining a sample that could reasonably be assumed to consist of mostly PBP tribal members. Including Shawnee County also allowed for the opportunity to include tribal members that had relocated to the largest nearby metropolitan area—Topeka—as a consequence of the relocation policy and need to seek employment. Such was the reasoning used to define the Prairie Band Community, more as a unit for analysis defined by the nature of the available data, as opposed to a sociocultural locality.

### **Appendix B References Cited**

Clifton, J.A. 1998. *The Prairie People: Continuity and Change in Potawatomi Indian Culture, 1665 - 1965*. Iowa City: University of Iowa Press.

Clifton, J.A. and Isaac, B. 1964. The Kansas Prairie Potawatomi: On the Nature of a Contemporary Indian Community. *Transactions of the Kansas Academy of Science* 67(1):1-24.

Edmunds, R.D. 1978. *The Potawatomis: Keepers of the Fire*. Norman, OK: University of Oklahoma Press.

Herring, J.B. 1988. *Kenekuk: The Kickapoo Prophet*. Lawrence: University of Kansas Press.

Hill, Edward E. 1981. *Guide to Records in the National Archives of the United States Relating to American Indians*. Washington DC: National Archives and Records Service, General Services Administration.

IHS (Indian Health Service) 2005. *The First 50 Years of the Indian Health Service: Caring and Curing*. Rockville, Md.: U.S. Department of Health and Human Services.

## **APPENDIX B**

### **DEFINITION OF THE “PRAIRIE BAND COMMUNITY”**

NARA (National Archives and Records Administration). 2009. Guide to Archival Holdings at NARA's Central Plains Region (Kansas City). <http://www.archives.gov/central-plains/kansas-city/holdings/rg-050-099.html>. May 15<sup>th</sup>, 2009.

Prairie Band Potawatomi Nation. 2011. Tribal Administration. Edited by S. Heck. <http://www.pbpindiantribe.com/tribal-administration.aspx>. November 29<sup>th</sup>, 2011.